

# **JOURNAL** *of the* **American Veterinary Medical Association**

*Formerly* AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Assn.)

H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

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Washington, D. C.

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## **PRESIDENT FOSTER ON THE ROAD**

President Foster has taken to the road. As this is written he is en route to the Pacific Coast to attend meetings in California early in January. As has been the case with all A. V. M. A. presidents in recent years, President Foster has received about twice as many invitations as he could accept. This applies especially to meetings held during the month of January. According to the information available, veterinary meetings, conferences and short courses will be held in about 24 different states this month.

The first meeting attended by Colonel Foster, as President of the A. V. M. A. was at Asheville, N. C., on October 21-22-23. This was the twenty-first annual meeting of the Southern States Veterinary Medical Association. A report of the meeting appears elsewhere in this issue of the JOURNAL. From Asheville, President Foster dropped down to Lakeland, Fla., to meet with the veterinarians of the Peninsular State on October 26-27.

The first of December found President Foster in Chicago, for a busy week. The Executive Board of the A. V. M. A. was in session for most of two days. The meeting of the United States Live Stock Sanitary Association overlapped one of these. Conferences and meetings of various committees followed in close

succession. Following custom, President Foster was an invited guest at the annual banquet of the National Association of B. A. I. Veterinarians, the evening of December 3. Upon leaving Chicago, he proceeded to Lincoln, Neb., to attend the annual meeting of the Nebraska State Veterinary Medical Association on December 8-9 and, incidentally, to look over the ground for the 1937 convention, to be held in Omaha. Thence he proceeded to Sioux Falls to meet with the South Dakota veterinarians.

While in California, President Foster will attend a meeting of the Veterinary Hospital Association of Southern California on January 2 and then go to University Farm, Davis, for the meeting of the California State Veterinary Medical Association and the University of California Veterinary Conference the following week. Working eastward, he will be in State College, N. M., for the meeting of the New Mexico Veterinary Medical Association, January 8-9. The next stop will be in Oklahoma City, January 11-12, for the meeting of the Oklahoma Veterinary Medical Association. For the next two days, President Foster will be in Wichita, for the annual meeting of the Kansas Veterinary Medical Association. Then back to Washington, for the inauguration ceremonies on January 20.

The last week in January will see President Foster at Columbia, Mo., for the meeting of the Missouri Veterinary Medical Association and the University of Missouri Veterinary Conference. Two days will be spent at Columbia and then he will proceed to East Lansing, Mich., to participate in the Michigan State College Short Course for Veterinarians. This event is scheduled for the entire week, but President Foster will not be able to reach East Lansing until the 27th. Tentative plans for February will include a trip to the South, to attend meetings at Auburn, Ala., and Baton Rouge, La., if a satisfactory schedule can be worked out. President Foster plans to attend the summer meetings of a number of state associations whose invitations to attend the winter meetings could not be accepted on account of conflicts of dates.

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### EXECUTIVE BOARD ELECTION

The special election in Executive Board District 10 (Michigan and Ohio) will come to a close on January 25. The voting has been unusually heavy, undoubtedly due to the popularity of the five candidates, all of whom are well known in the two states comprising the district. The five candidates include two college professors, an extension veterinarian, a practitioner and a state con-

trol official. The successful candidate will fill out the unexpired term of Dr. O. V. Brumley, president-elect of the American Veterinary Medical Association.

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### OMAHA CONVENTION DATES

At the meeting of the Executive Board of the American Veterinary Medical Association, held in Chicago, December 1, 1936, the dates for the 74th annual convention were fixed for

**August 16-17-18-19-20, 1937**

after hearing suggestions from the Committee on Local Arrangements and taking all important matters into consideration. Monday, August 16, will be set aside for the first session of the House of Representatives, the annual meeting of the Executive Board, and meetings of various committees. The opening session will be held on Tuesday morning, August 17.

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### A. V. M. A. STAFF AUGMENTED

It is with a great deal of pleasure that we announce the addition of Dr. E. C. W. Schubel, of Blissfield, Mich., to the full-time staff of the secretariat of the American Veterinary Medical Association. He comes in the capacity of assistant to the Secretary-Editor and has had a wealth of experience in several branches of the veterinary profession that should stand him in good stead in his new work. Thirteen years spent in general practice in Maryland, Indiana and Michigan have thoroughly acquainted Dr. Schubel with the various problems confronting the practicing veterinarian. For the past seven years he has been secretary of the Michigan-Ohio Veterinary Medical Association, one of the most flourishing local associations in the country, with a membership made up almost entirely of practitioners located in southern Michigan and northern Ohio. At the 1936 meeting of the Michigan State Veterinary Medical Association, Dr. Schubel was elected secretary, after serving for four years on the Board of Directors. One of the first things he did was to resume the regular publication of the *Michigan Veterinarian*.

For about eleven years Dr. Schubel was in commercial work, including the production of anti-hog cholera serum and sales work. In the latter capacity he visited all the states in the Corn Belt as well as the southern states, and in this way became acquainted with hundreds of veterinarians in this territory. Short

periods of employment by the State of Michigan and the City of Lansing gave him additional valuable experience in sanitary control work. It will be seen that Dr. Schubel has had a well-rounded experience in various branches of the profession that qualifies him to enter a new field that is limited only by the boundaries of veterinary activities. This field is constantly expanding and the boundaries, if there really be any, are continually being pushed back. We have only one national veterinary organization and in the future it will continue to serve all branches of the veterinary profession, just as it has in the past. With our augmented staff we hope to be able to do this job better than it has ever been done before.

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### PENNSYLVANIA WINS FIGHT AGAINST TUBERCULOSIS

Pennsylvania was declared the winner in a long fight against bovine tuberculosis on December 4, 1936, when the Keystone State took her place as the forty-third state on the accredited list. The event was celebrated in Harrisburg, on December 16, with appropriate "Achievement Day" ceremonies, attended by prominent veterinarians, public health officials and breeders.

In reviewing the history of bovine tuberculosis in this country, with particular reference to the efforts that have been put forth to gain control of the disease, Pennsylvania stands out prominently. Shortly after Koch announced his discovery of tuberculin, a Koch Lymph Commission was organized at the Veterinary Department of the University of Pennsylvania. The following extracts are taken from an announcement made by the late Dr. W. L. Zuill, quoted in an editorial entitled, "Tuberculin in Veterinary Medicine," in the *American Veterinary Review* for May, 1891:

The members of the Clinical Staff of the Veterinary Hospital of the University of Pennsylvania held a meeting on Saturday, February 14th, for the purpose of organizing a commission to examine by careful and scientific investigation into the value of Koch lymph in veterinary medicine. The commission intend to prosecute the study of this important question with the view of determining its value in three special directions. First: To determine whether or not it is of any value to the veterinarian in diagnosing tuberculosis in cattle. Second: To discover, if possible, what may be its value as a curative agent. Third: To prove, by a series of carefully made experiments, if it be possible to give healthy animals immunity to the contagion of tuberculosis, and for what length of time.

The Commission organized by the selection of Prof. W. L. Zuill, Chairman, and W. Edgar H. Landis, Secretary. The other members

of the Commission are Prof. Simon J. Harger, Drs. Chalkley H. Magill, Charles Williams, Wm. H. Ridge, Robert Formad and John Marshall.

The same announcement contained the following rather prophetic paragraph:

This investigation undertaken by the Commission of the Veterinary Department of the University of Pennsylvania to determine the influence of Koch's lymph in animals, is the first undertaken in this country. The Commission believe that this remedy of Koch's will prove to be of great value to the veterinarian and agriculturist by enabling them to detect the disease in its earliest stages, when it is so obscure as to evade the most careful physical examination. Should this prove to be true it will enable the stock owner to weed out the diseased stock from his herd even before they can do harm to his healthy cattle, or to the consumers of the products of his dairy.

It was in the following year (1892) that the first tuberculin test of a herd of cattle in America was made. This test was conducted on a farm near Philadelphia and the reactors were slaughtered for the purpose of verifying the accuracy of the test. In 1895, the Pennsylvania State Live Stock Sanitary Board was created and the late Dr. Leonard Pearson was appointed first state veterinarian of Pennsylvania. The following year the laboratory of the Board was established in Philadelphia, under the direction of Dr. Mazyck P. Ravenel. Extensive experiments were carried on here with a view to developing a method of vaccinating cattle against tuberculosis. In this work the late Dr. S. H. Gilliland took a prominent part. He succeeded Dr. Pearson as state veterinarian, following the death of the latter in 1909. Coöperative tuberculosis eradication work in the Keystone State was started in 1918, while Dr. C. J. Marshall was state veterinarian, but it was not until October 15, 1924, that the first county (Jefferson) was designated as a modified accredited area. The late Dr. T. E. Munce was then state veterinarian of Pennsylvania. It required twelve more years to clean up the heavily infected areas in the southeastern part of the state, Delaware, Lancaster and Montgomery counties finally yielding in the relentless campaign conducted against the bovine scourge.

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## APPLICATIONS FOR MEMBERSHIP

December kept pace with the four months immediately preceding, in the matter of applications for membership received. During the four months, August to November inclusive, 162 applications were received, an average of a fraction better than 40 per month. The month just closed was credited with 42 applications. These are given first listing this month, bringing the

total listed since the Columbus meeting up to 204. If this pace can be maintained during the next seven months, we will be within striking distance of 500 new members for the year. This number has been exceeded only once during the time that the present method of handling applications has been in effect.

So that our members may refresh their memories as to the manner of filing an application for membership, we are quoting Section 1 of Article 2 of the By-laws.

Application for membership shall be made upon a blank furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Application must be accompanied by the membership fee of \$5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Application must be filed with the Secretary and be examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application with name and address of applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary, as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary, and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

#### FIRST LISTING

- ACKERMAN, GRANT A. Hebron, Neb.  
D. V. M., Colorado State College, 1928  
Vouchers: E. C. Jones and Frank Breed.
- AICHELMAN, WILLIAM W. 829 N. Main St., Bellefontaine, Ohio  
D. V. M., Colorado State College, 1932  
Vouchers: R. Springer and C. E. Inskeep.
- ANDERSON, JOHN S. State House, Lincoln, Neb.  
M. D. C., Chicago Veterinary College, 1894  
Vouchers: Frank Breed and E. C. Jones.
- BARBEE, JAMES S. Sutton, Neb.  
D. V. M., Chicago Veterinary College, 1920  
Vouchers: S. M. Score and J. N. Barbee.
- BOLENBAKER, CAPT. ROGER F. 326 Brix Bldg., Fresno, Calif.  
D. V. M., Cornell University, 1916  
Vouchers: Col. A. L. Mason and L. G. Clark.
- CARLSON, FRED N. Faulkton, S. Dak.  
D. V. M., McKillip Veterinary College, 1918  
Vouchers: C. H. Hays and G. E. Melody.
- CLINE, MERTON L. Plainview, Neb.  
D. V. M., McKillip Veterinary College, 1917  
Vouchers: L. Collins and Frank Breed.

- COLLINS, WARNIE R. Bancroft, Neb.  
D. V. M., Iowa State College, 1931  
Vouchers: C. R. Collins and Darrell E. Trump.
- FOOTE, LT. BERNARD E.  
Hdqrs. Silver City Dist., C. C. C., Silver City, N. M.  
D. V. M., Kansas State College, 1934  
Vouchers: Maj. O. C. Schwalm and W. W. McMichael.
- GETZ, CAPT. AUSTIN T.  
Port of Embarkation, 58th St. & 1st Ave., Brooklyn, N. Y.  
D. V. M., Iowa State College, 1930  
Vouchers: Maj. Harry E. Van Tuyl and Herbert K. Moore.
- GIBSON, S. S. Randolph, Neb.  
D. V. M., McKillip Veterinary College, 1917  
Vouchers: Frank Breed and Floyd Perrin.
- GLENNEY, WILLIAM C. Tri-City Animal Hospital, Elgin, Ill.  
D. V. M., Iowa State College, 1936  
Vouchers: J. V. Lacroix and C. N. Bramer.
- HASSELBALCH, A. E. Saint Edward, Neb.  
D. V. M., Kansas City Veterinary College, 1912  
Vouchers: Frank Breed and Carl J. Norden.
- HERRING, LT. FRED L. United States Military Academy, West Point, N. Y.  
B. S., Franklin and Marshall College, 1931  
V. M. D., University of Pennsylvania, 1936  
Vouchers: Col. Robert J. Foster and Maj. E. M. Curley.
- HERSHBERGER, MAJ. FRANK C. Station Veterinarian, Fort Hamilton, N. Y.  
D. V. M., Kansas City Veterinary College, 1913  
Vouchers: Col. Robert J. Foster and Maj. George L. Caldwell.
- HILL, CAPT. VERNE C. 214 Meade Ave., Fort Leavenworth, Kan.  
D. V. M., Kansas State College, 1925  
Vouchers: Lt. Col. Jesse D. Derrick and Maj. Geo. J. Rife.
- LASSEN, LT. KEITH O. Dist. H., C. C. C., Fort Benning, Ga.  
D. V. M., Kansas State College, 1936  
Vouchers: Col. B. A. Seeley and J. L. Hopping.
- LEONHARD, ERNEST H. 334 Post Office Bldg., Oklahoma City, Okla.  
D. V. M., Colorado State College, 1936  
Vouchers: S. E. Douglas and L. J. Allen.
- LINDGREN, T. J. Edgar, Neb.  
D. V. M., Saint Joseph Veterinary College, 1916  
Vouchers: H. Gross and Frank Breed.
- LOVELL, MAJ. RAYMOND I. Presidio of Monterey, Calif.  
D. V. M., Ohio State University, 1914  
Vouchers: Col. A. L. Mason and Lt. T. C. Jones.
- LOY, CARL T. 1303 Drexel St., Omaha, Neb.  
D. V. M., Saint Joseph Veterinary College, 1923  
Vouchers: Harvey E. Smith and Rease Mitcham.
- LUNDBERG, FREDERICK O. Wausa, Neb.  
D. V. M., Chicago Veterinary College, 1917  
Vouchers: Frank Breed and Floyd Perrin.
- McEACHRAN, I. W. Geneva, Neb.  
D. V. S., Kansas City Veterinary College, 1905  
Vouchers: E. C. Jones and E. H. Meyer.
- MACE, LT. DON L. C. C. C. Hdqrs., Post Office Bldg., Redding, Calif.  
D. V. M., Kansas State College, 1935  
Vouchers: J. L. Heitt and Wm. C. Bellis.

- MAU, FRED C. 6003 S. Mozart St., Chicago, Ill.  
D. V. M., McKillip Veterinary College, 1918  
Vouchers: John Dickson and Israel Wallman.
- MAY, ARTHUR W. 4 Agassiz Park, Jamaica Plain, Mass.  
M. D. V., Harvard University, 1897  
Vouchers: H. W. Jakeman and Harrie W. Peirce.
- MILLER, ROY F. 309 Federal Bldg., Pierre, S. Dak.  
D. V. M., Kansas State College, 1935  
Vouchers: C. H. Hays and G. E. Melody.
- PERRY, HOWARD D. Yankton, S. Dak.  
D. V. M., Iowa State College, 1931  
Vouchers: C. H. Hays and G. E. Melody.
- PETERSON, LT. HAROLD O. 8217 19th Ave. N. E., Seattle, Wash.  
B. S., D. V. M., State College of Washington, 1936  
Vouchers: N. G. Covington and Hilton A. Smith.
- PHILLIPS, LT. HARRY L.  
5th C. C. Dist. Base, U. S. Army Wharf, New London, Conn.  
B. Sc., Pennsylvania State College, 1928  
V. M. D., University of Pennsylvania, 1935  
Vouchers: Norman J. Pyle and B. M. Lyon.
- ROSENWALD, ARNOLD S. 276 E. Seventh St., Winona, Minn.  
B. S., University of California, 1930  
D. V. M., Kansas State College, 1936  
Vouchers: R. R. Dykstra and John L. Meyers
- RYLAND, JOSEPH H. Box 514, Selma, Ala.  
D. V. M., Alabama Polytechnic Institute, 1923  
Vouchers: I. S. McAdory and F. P. Woolf.
- SANDIN, OSCAR Plattsmouth, Neb.  
D. V. M., Kansas City Veterinary College, 1912  
Vouchers: E. C. Jones and E. H. Meyer.
- SCHMELA, JOHN H. Norfolk, Neb.  
D. V. S., Kansas City Veterinary College, 1911  
Vouchers: S. M. Score and J. N. Barbee.
- SMITH, JOHN W. R. 1, Stockyards Station, Denver, Colo.  
D. V. M., Colorado State College, 1936  
Vouchers: S. E. Douglas and L. J. Allen.
- SPANGLER, DON H. 516 University, S. E., Minneapolis, Minn.  
D. V. M., Kansas State College, 1931  
Vouchers: Floyd Perrin, G. H. Leenerts and H. C. H. Kernkamp.
- STEIGLEDER, BEN H. 4525 Ellis Ave., Chicago, Ill.  
D. V. M., Cincinnati Veterinary College, 1916  
Vouchers: Israel Wallman and John Dickson.
- TAYLOR, IRWIN A. Durand, Wis.  
B. S., Iowa State College, 1933  
D. V. M., Iowa State College, 1935  
Vouchers: W. R. Winner and James S. Healy.
- THIELE, M. I. Prescott, Iowa  
D. V. M., Iowa State College, 1933  
Vouchers: Frank Breed, E. C. Jones and Chas. Murray.
- TUDOR, ALVEN O. 334 Post Office Bldg., Oklahoma City, Okla.  
D. V. M., Colorado State College, 1920  
Vouchers: S. E. Douglas and C. H. Fauks.
- VOLLMER, FLOYD A. Liberty, Ill.  
D. V. M., Ohio State University, 1936  
Vouchers: S. E. Douglas and L. J. Allen.

YOUNT, VIRGIL A. 334 Post Office Bldg., Oklahoma City, Okla.  
D. V. M., Colorado State College, 1936  
Vouchers: S. E. Douglas and L. J. Allen.

## Applications Pending

### SECOND LISTING

(See December, 1936, JOURNAL)

Applegate, Ralph W., 200 Morgan St., Tampa, Fla.  
Armfield, Frank P., Marianna, Fla.  
Bild, Charles E., 2635 N. W. 36th St., Miami, Fla.  
Brown, Lt. Robert J., Akron, Iowa.  
Clark, Andrew F., Box 617, Sarasota, Fla.  
Clarvoe, Harold M., 405 S. Howard Ave., Tampa, Fla.  
Dilts, Charles R., 5707 Nebraska Ave., Tampa, Fla.  
Gillis, DeWitt C., State Live Stock Sanitary Board, Tallahassee, Fla.  
Hamman, Capt. Fred I., Hdqrs. Casper Dist., C. C. C., Casper, Wyo.  
Hartzell, Lt. Harold P., Fort Douglas, Utah.  
Heishman, J. O., Station F, Jacksonville, Fla.  
Herman, Samuel E., 120 E. 59th St., New York, N. Y.  
Hoon, Lt. Henry R., 807 10th Ave., Lewiston, Idaho.  
Hughes, Harbard C., 532 W. Miami Rd., Jacksonville, Fla.  
Hyslop, Herman T., Burns & Co., Ltd., Prince Albert, Sask.  
Johnson, Raymond W., 1618 S. 24th St., Lincoln, Neb.  
Johnson, Samuel T., 816 Broad St., Jacksonville, Fla.  
Kennedy, Earl R., 3715 5th Ave., Moline, Ill.  
Lancaster, Lt. Harry R., Box 776, Tuscaloosa, Ala.  
McGinnis, Lt. Velmer W., Army Veterinary School, Army Medical Center, Washington, D. C.  
Martin, Walter D. Jr., 2635 N. W. 36th St., Miami, Fla.  
Miller, Herbert E., Box 404, Coral Gables, Fla.  
Morgan, Lt. Donald R., 402 E. 29th St., Vancouver, Wash.  
Omdalen, R. O., 6150 Greenwood Ave., Chicago, Ill.  
Reineccius, Lt. Jake L., Fort Snelling, Minn.  
Rippetoe, Lt. Culver W., Hdqrs. Missouri-Kansas Dist., C. C. C., Fort Leavenworth, Kan.  
Selkin, William J., 604 E. Gun Hill Rd., Bronx, New York, N. Y.  
Smit, Lt. Charles R., Fort Snelling, Minn.  
Smit, Lt. Walter, 230 5th Ave., Leavenworth, Kan.  
Spencer, Arthur H., Lake Worth, Fla.  
Tanner, Wallace J., 2117 4th St., Saint Petersburg, Fla.  
Thompson, Lt. William M., 1203 White Ave., Grand Junction, Colo.  
Todd, Lt. F. Arnold, Camp Charles M. Smith No. 11064, Waterbury, Vt.  
Utle, Thomas E., 1640 N. W. 14th St., Oklahoma City, Okla.  
Wann, Lt. Russell S., 2203 Jackson St., Alexandria, La.  
White, Lt. Alfred E. Jr., 633 Chester St., Glendale, Calif.  
Williamson, Arthur H., State Board of Health, Jacksonville, Fla.  
Wilson, Capt. Willis, Dayton, Wash.  
Winter, Edward F., 210 E. Hanna St., Tampa, Fla.  
Wirthlin, John R., 6002 Suwanee Ave., Tampa, Fla.  
Wiswell, Lt. Wilbur H., Veterinary Station Hospital, Fort Des Moines, Iowa.  
Zedlitz, Lt. Alfred C., 1203 8th St., Ballinger, Texas.

The amount which should accompany an application filed this month is \$10.00, which covers membership fee and dues to January 1, 1938, including subscription to the JOURNAL.

## COMING VETERINARY MEETINGS

- New Hampshire Veterinary Medical Association. State House, Concord, N. H. January 5, 1937. Dr. C. L. Martin, Secretary, University of New Hampshire, Durham, N. H.
- Pennsylvania, Conference for Veterinarians at University of. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. January 5-6, 1937. Dr. G. A. Dick, Dean, 39th St. and Woodland Ave., Philadelphia, Pa.
- California State Veterinary Medical Association and University of California Veterinary Conference. University Farm, Davis, Calif. January 5-8, 1937. Dr. Chas. J. Parshall, Secretary, Brentwood, Calif.
- Maine Veterinary Medical Association. Hotel North, Augusta, Me. January 6, 1937. Dr. S. W. Stiles, Secretary, Falmouth Foreside, Me.
- Cornell University, Annual Conference for Veterinarians at. New York State Veterinary College, Ithaca, N. Y. January 7-8, 1937. Dr. W. A. Hagan, Dean, Cornell University, Ithaca, N. Y.
- New Mexico Veterinary Medical Association. State College, N. M. January 8-9, 1937. Dr. T. I. Means, Secretary, Penn Road, Santa Fe, N. M.
- Ak-Sar-Ben Veterinary Medical Association. Elks Building, Omaha, Neb. January 11, 1937. Dr. J. N. McIlroy, Secretary, 3251 Leavenworth St., Omaha, Neb.
- Oklahoma Veterinary Medical Association. Skirvin Hotel, Oklahoma City, Okla. January 11-12, 1937. Dr. C. H. Fauks, Secretary, 1719 S. W. 15th St., Oklahoma City, Okla.
- Intermountain Livestock Sanitary Association. Ogden, Utah. January 11-13, 1937. Dr. D. E. Madsen, Secretary, Utah Experiment Station, Logan, Utah.
- San Diego County Veterinary Medical Association. San Diego, Calif. January 12, 1937. Dr. Donald E. Stover, Secretary, Zoological Research Bldg., Balboa Park, San Diego, Calif.
- Willamette Valley Veterinary Medical Association. Woodburn, Ore. January 13, 1937. Dr. Elwyn W. Coon, Secretary, Forest Grove, Ore.
- Kansas Veterinary Medical Association. Allis Hotel, Wichita, Kan. January 13-14, 1937. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kan.

New Jersey, Veterinary Medical Association of. Hotel Douglas, Newark, N. J. January 14-15, 1937. Dr. J. G. Hardenbergh, Secretary pro tem., c/o Walker-Gordon Laboratory Co., Plainsboro, N. J.

Ohio State Veterinary Medical Association. Deshler-Wallick Hotel, Columbus, Ohio. January 14-15, 1937. Dr. R. E. Rebrasier, Secretary, Ohio State University, Columbus, Ohio.

Idaho Veterinary Medical Association. Boise, Idaho. January 18, 1937. Dr. E. M. Gildow, Secretary, University of Idaho, Moscow, Idaho.

Wisconsin Veterinary Medical Association. Park Hotel, Madison, Wis. January 18-20, 1937. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. January 19, 1937. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

South Carolina Association of Veterinarians. Jefferson Hotel, Columbia, S. C. January 19, 1937. Dr. R. A. Mays, Secretary, 408-410 State Office Bldg., Columbia, S. C.

Indiana Veterinary Medical Association. Severin Hotel, Indianapolis, Ind. January 19-21, 1937. Dr. W. B. Craig, Secretary, 1420 N. Alabama St., Indianapolis, Ind.

Iowa Veterinary Medical Association. (Place not stated in notice.) January 19-21, 1937. Dr. C. J. Scott, Secretary, Knoxville, Iowa.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. January 20, 1937. Dr. L. E. Pike, Secretary, 1220 Bennett Ave., Long Beach, Calif.

Texas, State Veterinary Medical Association of. Fort Worth, Texas. January 20-21, 1937. Dr. Dee Pearce, Secretary, Box 335, Leonard, Texas.

Colorado Veterinary Medical Association. Albany Hotel, Denver, Colo. January 21, 1937. Dr. B. R. McCrory, Secretary, Colorado State College, Fort Collins, Colo.

Minnesota State Veterinary Medical Society. Hotel Saint Paul, Saint Paul, Minn. January 21-22, 1937. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Nevada State Veterinary Association. Reno, Nev. January 22, 1937. Dr. Warren B. Earl, Secretary, Box 1027, Reno, Nev.

Tennessee Veterinary Medical Association. Nashville, Tenn. January 25-26, 1937. Dr. A. C. Topmiller, Secretary, c/o Department of Agriculture, Nashville, Tenn.

- Michigan State College Short Course for Veterinarians. Michigan State College, East Lansing, Mich. January 25-29, 1937. Dr. Ward Giltner, Dean, Michigan State College, East Lansing, Mich.
- Missouri Veterinary Medical Association and Special Course for Graduate Veterinarians. University of Missouri, Columbia, Mo. January 26-28, 1937. Dr. C. L. Campbell, Secretary, 1817 Holmes St., Kansas City, Mo.
- Keystone Veterinary Medical Association. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. January 27, 1937. Dr. M. W. Allam, Secretary, Media, Pa.
- Massachusetts Veterinary Association. Hotel Westminster, Boston, Mass. January 27, 1937. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.
- Mississippi State Veterinary Medical Association. Tupelo, Miss. January 28-29, 1937. Dr. E. H. Durr, Secretary, Clinton Blvd., Jackson, Miss.
- Ontario Veterinary Association. Royal York Hotel, Toronto, Ont. January 28-29, 1937. Dr. H. M. LeGard, Secretary, 335 N. Main St., Weston, Ont.
- North Carolina State Veterinary Medical Association. Hotel Sir Walter, Raleigh, N. C. January 29, 1937. Dr. J. H. Brown, Secretary, Tarboro, N. C.
- Inland Empire Veterinary Medical Association. State College of Washington, Pullman, Washington. January 30, 1937. Dr. E. M. Gildow, Secretary, University of Idaho, Moscow, Idaho.
- Southern California, Veterinary Hospital Association of. Los Angeles, Calif. February 2, 1937. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.
- Connecticut Veterinary Medical Association. Hotel Bond, Hartford, Conn. February 3, 1937. Dr. Geo. E. Corwin, Secretary, State Office Bldg., Hartford, Conn.
- New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. February 3, 1937. Dr. R. S. MacKellar, Jr., Secretary, 329 W. 12th St., New York, N. Y.
- Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. February 3, 1937. Dr. Milton R. Fisher, Secretary, 3678 Dover Pl., Saint Louis, Mo.
- Houston Veterinary Association. Houston, Texas. February 4, 1937. Dr. D. B. Strickler, Secretary, 317 Federal Bldg., Houston, Texas.

Alabama Veterinary Medical Association and Short Course for Graduate Veterinarians. College of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala. February 8-13, 1937. Dr. I. S. McAdory, Secretary, Alabama Polytechnic Institute, Auburn, Ala.

Chicago Veterinary Medical Association. Palmer House, Chicago, Ill. February 9, 1937. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

Louisiana Veterinary Medical Association and Louisiana State University Veterinary Short Course. Dalrymple Memorial Building, Louisiana State University, Baton Rouge, La. February 9-10, 1937. Dr. C. M. Heflin, Secretary, Box 1933, Baton Rouge, La.

Hudson Valley Veterinary Medical Society. Poughkeepsie, N. Y. February 10, 1937. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. February 10, 1937. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Illinois State Veterinary Medical Association. Hotel Abraham Lincoln, Springfield, Ill. February 18-19, 1937. Dr. C. C. Hastings, Secretary, Williamsville, Ill.

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### STATE BOARD EXAMINATIONS

California Board of Examiners in Veterinary Medicine. University Farm, Davis, Calif. January 4-6, 1937. Dr. Nelson E. Clemens, Secretary, 183 Castro St., Hayward, Calif.

Connecticut State Board of Veterinary Registration and Examination. State Office Building, Hartford, Conn. January 5, 1937. Dr. Geo. E. Corwin, Secretary, State Office Bldg., Hartford, Conn.

Iowa Veterinary Medical Examining Board. State Capitol, Des Moines, Iowa. January 18-19, 1937. All applications must be in the office of the Division of Animal Industry not later than 8:00 A. M. on January 18. Further information may be obtained from the Secretary. Dr. H. A. Seidell, Secretary, State Capitol, Des Moines, Iowa.

South Carolina State Board of Veterinary Examiners. Jefferson Hotel, Columbia, S. C. January 19, 1937. Dr. W. A. Barnette, Secretary, Greenwood, S. C.

## IS STATE-WIDE MEAT INSPECTION POSSIBLE?\*

By WARREN P. S. HALL, Toledo, Ohio

*Division of Health*

This question is one that should hold much of interest to the veterinary profession and, in particular, it is one with which those who are engaged in meat inspection are at this time especially concerned. It must be answered, either in the affirmative or negative. However, at this time some explanation of the question must be given. To start with, the answer to this question can be given in the affirmative, but in the manner almost as does the small boy answer a question, "Yes—but." The small boy outgrows this type of answer usually and we must also, if we are to give an answer that becomes us. It is the second word of this answer that we must eliminate, and it can be. If we are ever to solve the problems of meat inspection, perhaps we must appreciate that we have grown up and not realized the fact. So let us see what constitute the "buts" in this answer.

In the first place, a different approach to the subject must be made, and as well a new conception of the problem must be developed, with a more logical evaluation of its importance as a public service, and a realization of its importance to the veterinary profession. The realization that meat inspection is a valuable public health activity through the lessening of disease, and that through inspection much benefit to the consumer has resulted. Also that this work of inspection has done much to advance the cause of disease eradication among cattle with the attendant benefits to the live stock industry, which is the background of the veterinary profession. These should be impressed upon the members of the profession. The veterinarian must be brought to a greater realization of the public service phases of veterinary activity.

Meat inspection is, because of its very nature, I believe, considered unquestionably as a field for the veterinarian, yet the attitude of the veterinary profession itself leads one to think that this new vision of this problem must be presented and developed. A few months ago, I was to speak at a veterinary association meeting and, as is usual at such meetings, the non-practice items were relegated to the end of the program. However, the speaker before me did unwittingly give the best introduction a speaker on meat inspection could have. This man had been discussing the subject of sheep diseases and in closing he described a particular

\*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

group of sheep that were doing so badly that he recommended slaughter as the best solution of the difficulty. It developed, however, that on the way to market the sheep became worse. So, as the speaker said, "a local butcher slaughtered them and no one lost anything."

I have a lot of respect for this speaker as a practitioner, yet I do feel that he is somewhat confused in his meat hygiene backgrounds, as are so many others. For some reason individual protection is recognized readily, yet regional protection is considered lightly. The recommending of the slaughter of sick animals is a common practice, we all know. However, the belief that by this practice no one loses, perhaps, is a fallacy. It may be that the veterinary profession, the live stock industry, the meat packer, and the consumer all lose because of its prevalence.

#### VETERINARY ASSOCIATIONS SHOULD HELP

So we have one of our "buts" in front of us, namely, the education of the veterinarian himself. This can be brought about only by greater interest on the part of veterinary associations in this work, and through the greater development of inspection courses in our veterinary schools and the training of those who are now in the field.

The various associations, whether national, state, or local, should place this subject in a far more prominent place than is customary at present. The placing of this branch of veterinary service as the last paper of a two- or three-day program is not at all in keeping with its importance as a veterinary problem. The realization that fields other than practice may present real contributions to veterinary medicine should be recognized. The fact is, that meat inspection will either remain a proper field for veterinary endeavor or will eventually pass into the hands of the sanitary engineer, the layman, or disintegrate into a purely political endeavor to repay the faithful.

Meat inspection, in some form, will persist, I feel sure. We are faced with the question of whether we as veterinarians are willing to let so vital a veterinary field go backward or stand by and, through lack of action, voice approval of the present state of non-federal inspection. The field of milk control already has drawn to it the layman and as well will meat inspection. Milk work should logically be a veterinary field. Analysis will show, however, that to a large extent the veterinarian has lost ground in milk control. Only through increased interest on the part of the veterinary groups will full realization of the importance of food control to the veterinary profession be brought out.

Progress has been made, along the lines above stated in promoting interest and giving opportunity to obtain knowledge, in some places. Michigan has begun an admirable attempt to educate the veterinarian in inspection work, a program that should be enthusiastically supported by all of us. For it is this type of effort that will go far in promoting interest and knowledge among those who are so necessary for the success of this program. Similar programs will do much to advance the cause and we must encourage their establishment.

#### CONSUMER MUST BE EDUCATED

A second "but" is that the education of the consumer and of the industry must be engaged in as well. Federal inspection is well established and the little purple stamp is accepted without question as a guarantee of safety. Too often, however, the consumer does not realize the fact that not all meats carry the B. A. I. legend.

The consumers should know that today only part of their meat is inspected by anyone. Approximately two-thirds of the nation's meat supply is under federal inspection; the remaining third is either inspected by municipal units or is not inspected at all. The amount of meat foods sold without benefit of inspection is large, and, due to the fact that inspection makes more difficult the slaughter of questionable animals, the supply without inspection becomes a more serious hazard to the consumer. We have some municipal inspection units which are operating but which, unfortunately, are of but little value, and we have a vast number of operators selling home-killed meats. In certain parts of the country we also have the exempted meat supplies with which to contend. This exempt meat supply can nullify any program that is established.

When the consumer becomes aware of these distinctions, the consumption of properly inspected supplies increases. I believe it can be demonstrated that plants, under proper inspection, realize an increase in their business due to increased consumer approval, where any sort of adequate education of the consumer is undertaken.

A program of publicity for the packing industry and the consumer would increase the demand for inspection, for both stand to benefit through its development. I would suggest, also, that the various breed associations can be interested in this project, for it is to their interest as well. These associations are fully aware of the benefits already received from the veterinary profession and would, I feel, be able to lend aid to this project.

Such a campaign would result in increased public demand and perhaps do away with the concept that this program is selfish, which might be the interpretation if promoted entirely by the veterinarian. We should realize that this program is vastly greater in importance than the benefit that we as veterinarians will ever receive.

#### CORRECT EXISTING WEAKNESSES FIRST

The third "but" is that before we can attempt to educate the consumer or the industry we must be willing to admit that our present system is fundamentally unsound and attempt to correct the weaknesses in our setup. The bad features should be recognized and admitted. The day of mutual admiration in this project is passed and we should face the facts. If they are sound, we should continue; if not, we should be willing to admit it and correct the fault.

The present state of meat inspection outside of the B. A. I. is certainly not efficient and lends itself to severe criticism when observed at all closely. Our present system of incoördinated control units spread about in our larger centers of population bring about a condition whereby much harm to sound inspection service occurs.

The inconsistencies of the various units within the state, as to regulations, qualifications, personnel, etc., are certainly not conducive of confidence on the part of the consumer or the industry. The prohibitions in one municipality do not apply in another and the restrictions so often put up as barriers to outsiders desiring to conduct business is almost in line with the idea of trade restraint. Possibly such restraints are necessary at present, but certainly they are a weak point in our program of extending protection. To me, it is a sorry state of affairs that requires such hurdles to be erected.

We have, in most states today a situation about as follows: A number of cities, towns, or counties set out to engage in a meat inspection program. Inasmuch as each unit is separate and distinct from every other, each unit, acting upon its own judgment, evolves a system of inspection. Regulations, methods, etc., are established and inspection begins. Whether or not the regulations are adequate is largely the result of chance in many cases. It might be added that the printed regulations are often far from the measures applied in the daily conduct of inspections. Advice is seldom asked for and then not often followed, it appears. Due to the fact that adjacent territory often has no inspection, it is usually deemed necessary to evolve restrictions. Unfortunately

the restrictions are often brought about by the pressure of those engaged in the industry whose ideas are warped by being impressed with the conception of an opportunity to restrict competition.

If a unit gradually evolves a satisfactory service, it is only through years of effort and after much difficulty that this occurs. In the meantime, however, much dissatisfaction has occurred and past events have a tendency to be brought up, somewhat as ghosts, to act as inhibiting forces. Too often, however, no progress is made. The work in the packing-house being usually less attractive than other duties included in the program, so frequently meat inspection suffers when other duties press. A study of municipal or local units will demonstrate these weaknesses. It is not at all difficult to find inspection units wherein inspection is conducted in direct conflict with established standards.

#### HONOR THE PURPLE STAMP

We should honor the purple stamp of inspection but only when it represents what it is supposed to represent. The stamping upon a carcass of "Inspected and Passed," when backed by competent postmortem inspection at the time of slaughter, is one thing, but the stamping of this legend on a carcass that has not been inspected at all is still another thing. Inspection forces exist today where the presence of the inspector is not considered necessary.

That a portion of our meat supply is beyond inspection limits is a phase of this problem that should be emphasized. Near every municipality, where suitable inspection standards are maintained, there develops a type of plant that is especially to be feared. These plants engage in the slaughter of animals which operators of houses under inspection will not buy, due to the probability of loss at the time of inspection. Remarkable as it seems, apparently these operators under inspection are in error, for no apparent losses do occur in these suspect animals.

It is this discrepancy between the losses that occur, for cause, under inspection, compared with the lack of condemnations without inspection, that should be of interest to the consumer and the man in the meat industry. It is quite logical, I believe, to assume that as great losses for cause should occur in this non-controlled group as are found under inspection. If the elimination of diseased meats is of value in protecting the consumer, its non-elimination surely must be harmful. At present, the consumer is defenseless, as is also the man in the industry; on the one hand,

through ignorance of the condition; on the other, because of irregularities in control under our present system.

Localization of control under the present setup is one of the great hindrances to advance. Through the establishing of a uniform state-wide program under one authority and with one set of standards, the program of inspection can advance rapidly and with it the attendant benefits. State-wide control should be patterned as closely as possible to Bureau standards and in this one step another great advance could be made. Perhaps one of the greatest weaknesses of our present setup is its lack of uniformity as to methods, dispositions, activities, etc.

It is realized that the question of correlating state and federal services brings up all sorts of opportunity for discussion, yet I feel that unquestionably greater effectiveness and coöperation would result from state-wide control, for even if complete correlation could not be obtained, a vastly greater uniformity would result.

This state-wide control should be centered in one department, and here let me state that milk control has almost exactly the same features to be corrected as has meat inspection. This central authority, whether the state veterinarian, state health department, the state department of agriculture or whatever its source, should have complete administrative authority.

#### CIVIL SERVICE NEEDED

Inspectors should be under civil service and of such caliber as to be able to carry out their duties properly—able to do their work not only because of ability but because of freedom from outside influence. I feel that this work is such that special qualifications are necessary and I cannot agree with the conception that so often is held that meat inspection activity is a desirable means of augmenting income. For this reason, I stress the matter of ability.

I believe that the exact working program will vary with the different states as conditions vary in these states. However in the main, agreement between federal and state organizations can be worked out.

The last "but" I want to present is this: "but we must get started on our program." The need for such a program is today very great and unless we do take action of a definite nature, it may soon be too late. It has been my observation that we are being displaced in inspection activities. I have noted the reduction of veterinary personnel in certain places with the increase in lay personnel. It has been publicly stated in Ohio, in refer-

ence to milk work, that the further away from milk control the veterinarian could be kept the better the control would be. This type of comment by a medical man is astonishing, if not illuminating. Recently a state-wide milk control bill was presented to the Ohio Legislature and it was disconcerting for some of us to discover that it was to be lay-controlled. I wonder if there will come a day when a similar bill for meat inspection will be presented. This last appears to be selfish, yet it is not wholly so, for meat inspection and milk inspection as well need the services that the veterinarian can give. No other group can supply the qualified men to undertake the responsibility, and it is a responsibility.

If these food control activities are allowed to fail, the protection that they can afford to the consumer, the live stock industry and the food industries as well must fail. We as veterinarians cannot permit this to come about. We have a responsibility in this matter that we must meet.

I would therefore encourage action on this question, definite and immediate action. If this is undertaken, we can answer the question proposed, "Is state-wide meat inspection possible?" with a definite *yes* without any qualifications.

#### Nebraska Membership in the A. V. M. A.

<i>Year</i>	<i>Members</i>	<i>Year</i>	<i>Members</i>
1923 .....	121	1930 .....	91
1924 .....	107	1931 .....	92
1925 .....	98	1932 .....	94
1926 .....	94	1933 .....	91
1927 .....	88	1934 .....	86
1928 .....	82	1935 .....	76
1929 .....	89	1936 .....	92
1937 .....		??	

#### World's Dairy Congress in Berlin

The Eleventh World's Dairy Congress will be held in Berlin, Germany, August 22-28, 1937. Former Congresses, which are held under the auspices of the International Dairy Federation, have been held as follows: Brussels (1903), Paris (1905), The Hague (1907), Budapest (1909), Stockholm (1911), Bern (1914), Paris (1926), London (1928), Copenhagen (1931), Rome and Milan (1934).

## SOME OF THE PATHOLOGICAL LESIONS ASSOCIATED WITH INDIGESTION IN DOGS\*

By LEONARD W. GOSS, *Columbus, Ohio*

*Department of Veterinary Pathology, Ohio State University*

The diseases of the digestive tract of the dog are brought to the attention of the veterinarian more frequently than any other group of diseases. These may be primary, but in many cases they are secondary to other disease conditions or complications associated with some other disease.

The most common primary cause is the diet, and probably the most common condition to which it is secondary is parasitic infestation, and following this is distemper. The average dog-owner has but little knowledge of the food requirements for the dog. Consequently he is inclined to feed the dog such food as he likes rather than those foods which are properly balanced in the essentials of a good ration. At the present time, the commercial dog food situation is in such a chaotic condition that he gets but little if any assistance. Consequently the diet is frequently at great fault and a common cause of disease.

Parasitic infestation is so common to young puppies and to many older dogs that consequently this condition is one of the most common causes of indigestion. Nor is the situation improved by the indiscriminate use of commercial worm medicines which are frequently used in excessive amounts and when they are not indicated. Enteritis is a common complication with other diseases which may be of an acute or chronic nature. Accidental or intended administration of drugs which are poisonous in the quantity received are responsible for some cases of enteritis.

Practically all auto-intoxications are associated with digestive disturbances.

Much could be said regarding the causes of the diseases which involve the digestive tract. Like other inflammations, there is an irritant of mechanical, physical or chemical nature which is responsible for the lesion. The extent to which it develops is largely dependent upon the resistance of the individual animal and the activity of the causative factor.

Goldberger<sup>1</sup> and his coworkers have done considerable work on diseases of the digestive tract under the name of "black tongue." Sebrill<sup>2</sup> shows that there is a fatty degeneration of

\*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

the liver and kidneys as the result of deficient diet. Woolridge<sup>3</sup> states that catarrhal enteritis is by far the most frequent form of enteritis in the dog. Wright,<sup>4</sup> in his discussion of the gastric and intestinal diseases, calls attention to other disease conditions which are frequently ascribed to disease of the digestive tract, and that gastritis is a much abused term.

#### CATARRHAL ENTERITIS FREQUENTLY MET

The observation of over 6,000 necropsies on dogs has revealed that a large proportion of these dogs show catarrhal enteritis. This may be the primary disease of a secondary contributing factor in the death of the animal. The seriousness of catarrhal enteritis is frequently underestimated. The symptoms associated with this condition have a great many variations. This probably is due to the varying extent of the disease, the complications, or to the disease conditions to which the enteritis is secondary.

Because of the frequency of catarrhal inflammation of the dog's digestive tract, this discussion will be confined largely to this condition. Various causes having lowered the resistance within the digestive tract, the bacteria which are there in large numbers are permitted to multiply within the ingesta. Under these conditions the volume of ingesta and exudate are present in increased amounts. Through the activity of the existing bacteria and those introduced from the exterior, a relatively large amount of material is produced which is toxic. This chemical substance acts upon the single layer of columnar epithelium which lines the intestinal tract covering the villi and extending into the tubular glands.

The cells at first undergo cloudy swelling. This is followed by further degeneration, death and desquamation (fig. 1). The extent to which this desquamation goes will vary with the intensity of the cause, the resistance of the animal and other contributory factors. With this condition there is exudation of leukocytes and plasma and in some instances considerable numbers of red blood-cells in the case of hemorrhagic complications. The exudation process is largely for the purpose of diluting the irritant. The local glands also take an active part by the secretion of mucous and serous fluids. The desquamation of the epithelium begins at the free end of the villi and extends toward the base in proportion to the degree of irritation.

The loss of this epithelium allows the absorption of the toxic substance formed by the bacterial action and other substances which are toxic but under normal conditions are prohibited from

being absorbed, by the protective action of the epithelium. As numerous capillaries are just beneath the epithelial layer, they are exposed when it is destroyed. Then absorption takes place by osmosis and diffusion as the surface exposed may be very extensive. A large amount of fluid toxic to the individual is disseminated over the body by the blood-vessels and lymphatics. This



FIG. 1. Cross section of the small intestine showing the desquamation of the epithelium of the villi.

brings about intoxications which account for many of the various symptoms exhibited. These substances are carried by the blood-stream indirectly to the liver where they cause cloudy swelling and if continued will result in fatty degeneration and indurations of the liver. The latter in turn may be responsible for ascites and other symptoms characteristic of disease of the liver.

#### KIDNEY DISTURBANCES

The kidneys, being one of the chief avenues for the elimination of waste products from the body, receive much of the toxic substance from the blood where it causes degeneration, within the cells of the tubules of the kidneys. This is recognized by swelling

of the epithelium, vacuole formation, followed by desquamation with the formation of granular and hyaline casts and the presence of albumin in the urine. This may be followed by indurations, occlusion of some of the tubules and cystic formation. It is fortunate that the kidneys have a large reserve capacity which allows for urinary function even after considerable portions of the kidneys have been damaged.

#### HEART BECOMES INVOLVED

The toxic products absorbed from the intestinal tract also cause albuminous degeneration of the cardiac muscle. This impairs the function of the heart, by weakening its force and slowing the circulation of the blood. The impaired circulation and the presence of the toxic products in the blood-stream bring about general passive congestion and edema of the lungs. If the condition is chronic, catarrhal bronchitis is prone to develop.

Chronic catarrhal enteritis, by its intoxicating effect, is responsible for many of the diseases of the skin, and is an influencing factor with regard to the degree of disturbance associated with sarcoptic and dermadectic infestations.

In addition to the fluid toxic substances which are absorbed from the intestinal tract, large numbers of bacteria may enter the unprotected capillaries and be disseminated to all parts of the body where they may lodge and be responsible for endocarditis, pleuritis, purulent nephritis and various other metastatic abscesses. Many aged dogs which have had chronic enteritis show a marked indurative nephritis, and cirrhosis of the liver. After these conditions have progressed until these organs are no longer capable of conducting their functions, there is a probability that the case will be diagnosed as nephritis, hepatitis or valvular insufficiency, dependent upon which organs are involved to the greatest extent.

With regard to the symptoms observed by the clinician and the lesion present, one rather important thing is that gastritis, as indicated by the extent of visible inflammation of the stomach, is quite rare as compared with the number of times it is incriminated. This may be due to the frequent occurrence of vomiting with such conditions as enteritis, distemper, intussusception, occlusion of the intestine, disease of the liver, disease of the kidneys, pyometra, tumors in the abdominal cavity, calculi of the urinary tract, and additional conditions which may involve the cerebral centers concerned in vomiting.

## SEVERE HEMORRHAGIC ENTERITIS

Severe hemorrhagic inflammations of the digestive tract are at times encountered. There seems to be no specific cause. There are, however, indications that the cause is of a more intense irritation than that which produces catarrhal inflammation. The degree of destruction to the epithelium of the intestinal tract, with the high state of congestion of the blood-vessels and the large amount of blood which passes from the mucous membrane, also the associated stomatitis characteristic of this condition, would indicate that the irritant is intense and has progressed to a point that corrections are in many cases impossible. It frequently seems to be of sudden onset but may be the acute termination of a chronic condition. The latter must be true if it is caused by vitamin deficiency, as is the belief of Goldberger<sup>1</sup> and others.

The seemingly moderate lesions associated with catarrhal enteritis, while small at any one place, are very extensive, which accounts for the absorption of a large amount of material with a far-reaching effect. In addition to the intoxication which is taking place, there is much disturbance in the preparation of food for absorption through its decomposition. Also its absorption is greatly interrupted and the tissues of the body are also put in a condition which makes it difficult to utilize nourishment when brought to them by the blood-stream. The exudation into the digestive tract and the associated diarrhea bring about a marked dehydration.

## TREATMENT

The pathological lesions indicate that in the treatment due consideration must be given to the removal of the cause. Drastic purges should not be resorted to, as they will remove more of the already degenerating epithelium and increase the existing dehydration, thus aggravating the condition. Substances should be administered which will protect the inflamed mucous membrane. The tissue dehydration should be corrected. The diet should be rich in the essential food elements and be in a form that will be readily assimilated.

## REFERENCES

<sup>1</sup>Goldberger, J., Tanner, W. F., and Sayer, E. B.: A case of black tongue with postmortem findings. U. S. Pub. Health Rpt. (Nov. 16, 1923.)

<sup>2</sup>Sebrill, W. H.: Fatty degeneration of the liver and kidneys in the dog apparently associated with diet. U. S. Pub. Health Rpt., xlii (Nov. 8, 1929), no. 45.

<sup>3</sup>Woolridge, G. H.: Encyclopedia of Veterinary Medicine, Surgery and Obstetrics. Vol. I. Veterinary Medicine. (Henry Frowde and Hodder & Stoughton, London, 1923.)

<sup>4</sup>Wright, J. G.: The diagnosis of gastric and intestinal disease of the dog and cat. Proc. 50th An. Cong. (1932) Nat. Vet. Med. Asso. Gr. Brit. & Ire.

## DISCUSSION

DR. PETER OLAFSON: In discussing this interesting paper I want to emphasize two or three points.

In middle-aged and old dogs, many cases of vomiting and dehydration are due to uremia. Acute or chronic interstitial nephritis terminating in uremia causes symptoms that are commonly diagnosed as gastritis.

The stomatitis of uremia has been confused with black tongue described by Goldberger. In our locality, nutritional black tongue is extremely rare if it occurs at all. Cases that have been so diagnosed have not responded to yeast or liver therapy. Many of them have proven to be uremia.

Our autopsy work supports the point made by Doctor Goss in regard to the harm that may result from excessive or improper use of worm medicines.

I have no information on the relationship of enteritis to nephritis. There is a great difference of opinion in regard to the effects produced by various diets. Some think that a pure meat diet is the cause of kidney damage, while others are equally certain that the cereal diets are harmful. Extensive work on the relationship of various diets to the health and life span of dogs would be interesting.

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Bureau Transfers

DR. CHARLES BEVERLY (Cin. '06), from Fostoria, Ohio, to Detroit, Mich., on meat inspection.

DR. W. C. DYE (K. C. V. C. '08), from Kansas City, Mo., to San Antonio, Texas, on field inspection.

DR. FRED STORZ (K. S. C. '32), from Sacramento, Calif., to Chicago, Ill., on meat inspection.

DR. EDWIN L. PECK (K. C. V. C. '13), from DeSoto City, Fla., to Omaha, Neb., on stock yards inspection.

DR. D. D. TIERNEY (McK. '08), from Austin, Minn., to South Saint Paul, Minn., in charge of meat inspection.

DR. L. E. EPPLE (O. S. U. '08), from Mason City, Iowa, to Austin, Minn., in charge of meat inspection.

DR. E. M. BERROTH (K. S. C. '20), from Kansas City, Kan., to Mason City, Iowa, in charge of meat inspection.

DR. W. O. NEY (K. C. V. C. '07), from Columbia, S. Car., to Knoxville, Tenn., on meat inspection.

DR. HARRY F. KERN (Colo. '11), from San Juan, P. R., to San Antonio, Texas, on field inspection.

DR. WALTER W. SHARTLE (Ind. '09), from Baltimore, Md., to Chicago, Ill., on meat inspection.

DR. JESSE L. SHABRAM (K. C. V. C. '17), from Fort Worth, Texas, to Kansas City, Mo., as associate live stock market specialist, Packers and Stockyards Division.

DR. GROVER C. GULICK (K. C. V. C. '16), from Marshalltown, Iowa, to Oklahoma City, Okla., on meat inspection.

DR. HARRY E. SKOOG (K. S. C. '30), from Pittston, Pa., to Smithfield, Va., on meat inspection.

## OBSERVATIONS ON CANINE BABESIASIS (PIROPLASMOSIS)\*

By D. A. SANDERS, Gainesville, Fla.  
Florida Agricultural Experiment Station

In 1895, Piana and Galli-Valerio<sup>1</sup> announced that a disease known as biliary fever or malignant jaundice of hunting dogs in the province of Lombardy, Italy, was due to the presence of a piroplasma in the blood which they named *Pyrosoma bigeminum* var. *canis*. This organism was subsequently designated *Piroplasma canis* and later *Babesia canis*. The disease and its etiological agent was observed by workers in France, Russia, England, Africa, India, China, the Philippines and other places. From these reports it is evident that canine babesiasis is widely distributed over the world, being especially prevalent in tropical zones where the acute form of the infection is often reported. In subtropical and temperate regions, canine babesiasis is considered to be less frequent and to assume the nature of a chronic infection.<sup>2</sup> In 1914, this disease was recognized by Martinez<sup>3</sup> on the island of Puerto Rico and in 1918, by Clark,<sup>1</sup> in the Panama Canal Zone. In 1934, Eaton<sup>4</sup> observed intra- and extracorporeal organisms in the blood of a dog in Jacksonville, Florida, which were identified by Mayne as *Babesia canis*. This was the first report of canine babesiasis in the continental United States.

Dr. J. H. Yarborough, of Miami, Florida, has encountered, during the past several months, a debilitating malignant disease of dogs, especially greyhounds, in various kennels of the lower east coast of Florida, in which affected animals exhibited clinical manifestations different from any condition observed during previous years. Affected greyhounds were incapacitated over prolonged periods, during which time their training and coursing activities were interrupted. Symptoms manifested by greyhounds and other breeds under observation were often somewhat vague, but in typical untreated cases, which often extended over a period of weeks, there occurred varying degrees of weakness, inappetance, vomiting, anemia, pallor of the mucous membranes, slight icterus of the sclera, intermittent fever, mucopurulent eye discharge, enlargement of the spleen and, less often, a cough, this latter condition being more pronounced when the animals were on the lead.

Hematological examinations of these cases showed a decrease in the number of erythrocytes, a varying degree of leukocytosis

\*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

of the polymorphonuclear variety, a low hemoglobin content, and in advanced stages, a pale watery condition of the blood. Untreated dogs which died in the course of a few weeks sometimes developed jaundice towards the end and the urine of such animals exhibited a greenish or reddish tinge. These symptoms led to a tentative diagnosis of canine babesiasis. Moreover, specimens of the brown dog tick, *Rhipicephalus sanguineus*, were usually taken from affected animals and these cases responded favorably to nursing and energetic treatment for babesiasis. A definite diagnosis, however, was not immediately possible, owing to the fact that the causal organism was not observed in carefully prepared blood-films from affected animals. Among numerous stained smears examined only one instance was recorded where a suspicious intracorpuseular pear-shaped body was noted and it was considered that sufficient evidence, based on blood examinations, was not at hand to warrant a definite diagnosis of this chronic disease.

In order to make a more detailed study of this condition, a greyhound which had become weak and unthrifty during the 1936 racing season was selected as a typical case representing the condition and shipped to the veterinary laboratories in Gainesville. Upon arrival, this animal presented, in addition to a weakened and unthrifty appearance, paleness of the mucous membranes, suppressed appetite, vomiting, slight intermittent fever, a cough and enlarged spleen. Examinations showed a hemoglobin content of 9.0 gm per 100 cc of blood on the Dare instrument. There was present a polymorphonuclear leukocytosis and a decreased number of erythrocytes per volume of blood. A few adult specimens, mostly males, of *R. sanguineus* were taken from the animal. Blood-smears stained by Wright's or Giemsa's method failed to show *Babesia*.

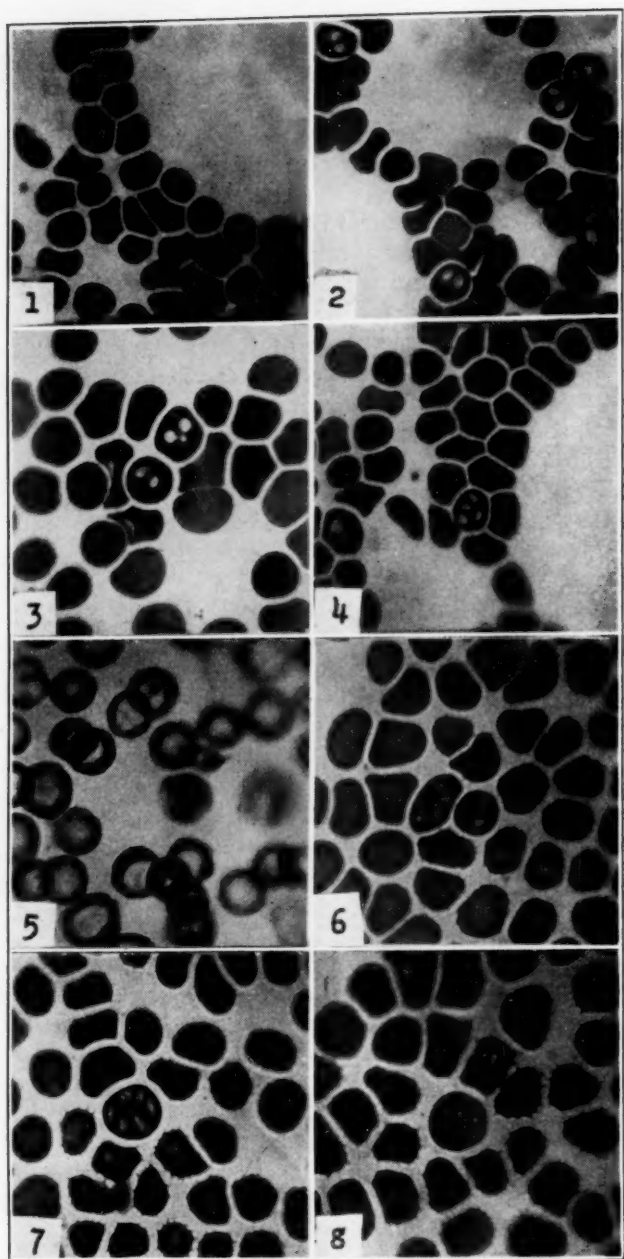
Following this preliminary examination, a local healthy pup was given an intraperitoneal injection of 10 cc of blood from this greyhound. On the fifth day following this injection, the test pup exhibited loss of appetite, malaise and elevation of temperature. Carefully prepared and stained blood-films showed the presence of a few *Babesia*. During the following four days, the above symptoms were aggravated, the hemoglobin content of the blood had been reduced from 16 to 8.2 gm per 100 cc (Dare). However, the hematozoön parasites were never very numerous in the peripheral circulation. Blood-counts showed the presence of a polymorphonuclear leukocytosis and oligocythemia in the test pup. On the sixth day following the initial reaction, it was difficult to demonstrate the parasites in carefully prepared blood-

films of this test pup, *Babesia* of doubtful identity being observed only rarely and by careful search.

It has been shown by workers<sup>5-10</sup> that the spleen, with its reticulo-endothelial system, has a distinct bearing on the course of some hematozoön infections in man and animals in that this organ assists in acquiring and maintaining immunity. Animals apparently in perfect clinical condition but harboring latent infections suffered a severe relapse when splenectomized, while animals not harboring such infections developed no important sequelae as a result of the operation. The activation of these latent infections by extirpation of the spleen resulted in flooding the peripheral circulation of the carrier or premunized animal with the hematozoön agent. Under these conditions the microparasites were readily demonstrated in the blood by staining methods.

The test pup that had been inoculated twelve days previously with blood from the affected greyhound, and from which blood-smears showed the organism to be exceedingly scarce, was splenectomized for the purpose of obtaining conclusive evidence, based upon blood examinations, of the presence of babesiasis. The operation of splenectomy was performed under general anesthesia. An incision was made in the region of the upper anterior left flank, parallel with and a sufficient distance posterior to the last rib for placing sutures. Sterile gauze placed over the incision was slit and the peritoneal edges sutured thereto. The peritoneal incision was gradually enlarged as the sutures were extended. The spleen, which was several times its normal size, was observed lying in position between the stomach and left kidney ventral to the lumbar transverse processes. The enlarged organ, together with its omental attachment, was gradually withdrawn. The splenic attachment was clamped, blood-vessels ligated, and the organ removed, after which the omental stump was placed in the abdominal cavity and the operative incision sutured.

Twenty-four hours following the splenectomy, carefully prepared blood-smears stained by Wright's or Giemsa's method showed the presence of hematozoön, pear-shaped parasites, *Babesia canis* (Piana and Galli-Valero 1895). (See figs. 1 to 9.) These typical pear-shaped forms, pointed at one extremity and rounded at the other, were 4.5 to 5 $\mu$  in length. Variations from these measurements occurred, depending largely upon the number parasitizing a single erythrocyte. *B. canis* was subsequently observed in a great variety of forms, including round, polygonal, pear-shaped, vacuolated, budding, marginal and other



FIGS. 1 to 8. Photomicrographs showing intracorporeal forms of *Babesia canis* occurring in the blood of a splenectomized pup.

reproducing phases. In many instances multiple infections of from four to 16 individuals were observed within a single erythrocyte. They occurred frequently in pairs and in this case a string of granules was often observed connecting the two parasites at their pointed extremities.

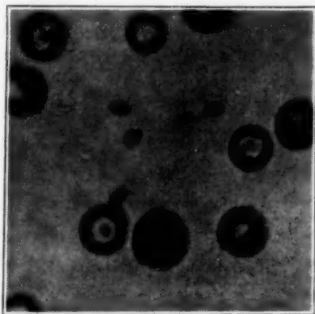


FIG. 9. Photomicrograph showing extracorporeal forms of *Babesia canis* in the blood of a splenectomized pup.

Many erythrocytes containing several organisms were observed as faint outlines, being devoid of hemoglobin and apparently undergoing dissolution as a result of the parasitic invasion. Groups of four to eight pear-shaped microparasites were often observed in the plasma, indicating their recent escape from an erythrocyte. A polymorphonuclear leukocytosis, polychromatophilic degeneration of the erythrocytes, marked anemia, poikilocytosis, anisocytosis, erythroblasts and other characteristic findings of severe hematozoön infections were observed in the splenectomized test pup. As a result, the hemoglobin content of the blood was so depleted that it failed to register on the hemoglobinometer.

The animal died on the sixth day following splenectomy. Splenectomies of additional test animals confirmed these observations, and it was found that puppies were more susceptible to this test, other factors being equal, than older dogs. Puppies splenectomized during or shortly following clinical reactions usually succumb within 24 to 48 hours, showing *B. canis* in numerous forms. Such splenectomized pups have frequently been observed to eat a hearty meal after recovery from the anesthesia and before prostration ensued from the severe parasitic attack.

On May 3, 1936, Dr. John R. Wells injected a pup at West Palm Beach, Florida, with 6 cc of blood taken from a dog showing symptoms and postmortem lesions of malignant jaundice. On the same day, this pup was taken to Gainesville by the writer

and held under observation in isolation quarters. On the fifth day following the injection, the test pup was depressed, failed to eat and exhibited a noon temperature of 104.8° C. As the teeth were erupting at this time, no special significance was given these physical irregularities until, upon making a blood-count, a total of 17,775 leukocytes per cubic millimeter of blood was registered. By careful search of stained blood-films, an occasional *Babesia* was observed parasitizing the erythrocytes. The temperature remained elevated for 48 hours and the hemoglobin content of the blood was lowered approximately 15 per cent. An occasional parasite was observed for a period of four days. The pup, in the meantime, recovered.

During July, 1936, a registered pointer showing grave symptoms of malignant jaundice from Micanopy, Florida, was presented to Dr. C. A. Palmer's veterinary clinic in Gainesville. Blood examinations failed to reveal the presence of *B. canis*. Fifteen cc of venous blood was secured from this animal and given intraperitoneally to a year-old local dog. No clinical or microscopic evidence of babesiasis developed in the test dog. This test dog was splenectomized on the 13th day, whereupon a state of chronic babesiasis ensued. In the meantime, the pointer which had received energetic treatment for babesiasis recovered its pristine condition and was dismissed from the hospital on the tenth day after admittance. These results led to the belief that the test animal was harboring a latent infection of *B. canis* prior to injection of blood from the pointer and was, therefore, not susceptible to a super-imposed infection. While it is not claimed that all cases of jaundice are due to *Babesia* infection, yet it appears that many cases of so-called yellow jaundice encountered in the South are associated with *B. canis*.

Pups receiving injections of small quantities of blood from recovered dogs frequently develop a mild clinical reaction without elevation of temperature, showing only a few microparasites occurring for a short period in the blood. Young susceptible pups which receive injections of blood from splenectomized dogs showing a rich parasitic invasion of the erythrocytes by *B. canis* usually develop acute reactions after an incubation period of four to seven days, depending upon the amount of inoculum employed. In table I will be found an example of the observations recorded on a fatal case thus inoculated.

#### DIAGNOSIS

The acute form of canine babesiasis offers no particular difficulty in making a diagnosis. The high temperature, increased

TABLE I—Reaction of pup to injection of blood from splenectomized carrier of canine babesias.

DATE (1936)	TEMPER- ATURE (° F.)	PULSE	RESPIRA- TIONS	HEMOGLOBIN CONTENT PER 100 CC OF BLOOD (16 GRAMS—100%)		CORPUSCLES PER CMM BLOOD		BABESIA DEMON- STRATED IN BLOOD	REMARKS
				Gm	%	Reds	Whites		
7/1	101.0	104	Frequency increased	16	100	9,000,000	10,000	—	Recipient given 10 cc blood intraperitoneally
7/3	102.4	100		15.2	95	8,750,000	12,500	—	Mucopurulent eye discharge, depressed. Pup seeks se- cluded place
7/4	103.8	130		14.4	90	7,500,000	13,000	—	Blood infective for susceptible pup
7/5	104.9	160		8.0	50	5,000,000	17,500	++	Polychromatophilic degenera- tion of erythrocytes. Aniso- cytosis and presence of er- ythroblasts
7/6	106.6	—		4.8	30	3,500,000	20,000	+++	Polymorphonuclear leukocytes predominate. Pokilocytosis
7/7	98.0	—		1.6	10	1,980,000	—	+++	Death

pulse and respiration, progressive anemia, jaundice, the history of tick infestation, and the demonstration of the causal organism are usually observed. It should be remembered, however, that the ticks which transmit the disease while engorging may detach and drop from the animal several days before clinical symptoms of babesiasis are manifest.

The chronic form of canine babesiasis which has been encountered in Florida and in which the presence of the causal organism is by no means easy to demonstrate, offers more difficulty of diagnosis. In these cases the symptomatic manifestations, together with the hematologic findings, are of value in making a diagnosis. The chronic cases usually have a low hemoglobin content per volume of blood, they exhibit anemia and pallor of the mucous membranes, loss of condition, weakness, often show intermittent fever and the presence of ticks. In later stages, jaundice is sometimes observed and in this case hemoglobinuria or biliruria may occur.

Examination of these chronic cases shows a polymorphonuclear leukocytosis, reduced number of red cells and a pale, watery condition of the blood. Microscopic examinations of the blood usually fail to reveal the presence of *Babesia*. Nutritional anemia and anemia due to intestinal parasites and leukocytosis due to other causes must be considered in the findings. The mucopurulent eye discharge and cough which may be associated with this condition are to be differentiated from distemper infection. The coexistence of many of these conditions which complicate the diagnosis must be considered.

In those locations where there is doubt of the existence of canine babesiasis, the inoculation of a susceptible pup will often be sufficient for making a diagnosis. As a further means of diagnosis, the test pup may be splenectomized and, in case babesiasis is present, the microparasites in various stages of development will be found in peripheral blood-smears, both free in the plasma and parasitizing the red blood-cells.

Postmortem examinations of dogs which succumb to the disease show the internal organs to be icteric and the blood pale and watery. The spleen is enlarged and the splenic pulp soft. The kidneys are congested and the urinary bladder distended with urine containing blood-coloring matter. The liver is enlarged and yellow, and the gall-bladder distended with dark, thick bile. The intestinal contents are often bile-stained, the feces being of a bright yellow color. Petechial hemorrhages often occur on the epicardium, endocardium and pleura. Lesions of bronchopneumonia are often observed.

## TRANSMISSION

Natural vectors of canine babesiasis have been found to be various species of ticks. The brown dog tick, *R. sanguineus*, which is a cosmopolitan species, has been incriminated as the transmitting agent in many countries. According to Wenyon,<sup>1</sup> this species is undoubtedly responsible for transmission in Asia, Europe and North Africa, and was found by Christophers<sup>2</sup> to be responsible for the transmission of the disease in India. The infection is hereditary, being transmitted through the egg of sexually-mature female ticks that imbibed blood from infected or premunized dogs. The larval stage is not capable of transmitting the causative agent but the subsequent nymphal and probably the adult stages are capable of transmitting the disease.

*R. sanguineus* is by far the most common species encountered in kennels and on animals affected with canine babesiasis in Florida. This tick is well established throughout Florida and the southern half of Texas. It is also continuously present in the larger cities in north Texas and in those of the Gulf Coast states. It has been found infesting premises in cities in Arizona, Illinois, Indiana, Maryland, Missouri, New Jersey, New York, Ohio and Pennsylvania and in the District of Columbia. Local infestations may occur wherever dogs are transported from the infested area of the southern states; such infestations, if established in heated buildings in the North, may persist indefinitely.<sup>11</sup>

*Ixodes ricinus* was designated the transmitting agent in Italy. A closely related species, *I. ricinus* var. *scapularis*, has a wide distribution in the United States and appears to have been collected from Maryland south to Florida, and west to Louisiana and Texas.<sup>12</sup> Another closely related species, *I. californicus*, also occurs in California.

The Rocky Mountain spotted fever tick, *Dermacentor andersoni* (venustus), has been found by Brumpt and Larrousse<sup>1</sup> to be capable of transmission of canine babesiasis. The infection imbibed by adult female ticks engorging on infected animals is transmitted through the egg, larvae and nymphal stages and is inoculated into susceptible dogs by the subsequent adult stage. This tick is distributed throughout the Rocky Mountain region in the states of Montana, Wyoming, Colorado, Utah, Idaho, northern Nevada, eastern Washington, eastern Oregon and northeastern California. It occurs also in limited areas in northern New Mexico and in western South Dakota. The tick

is commonly found in the Canadian provinces bordering on the infested areas in the United States.<sup>11</sup>

Another species present in the United States and quite common in Florida, *D. variabilis*, may also be incriminated in the transmission of canine babesiasis. Much research will be necessary before definite statements can be made regarding the rôle of each of these and other external blood-sucking parasites in the transmission of canine babesiasis.

It is to be remembered that recovered dogs are in a state of relative immunity or premunition and although perfectly healthy continue to harbor the infection for months after clinical recovery and apparent disappearance of the parasites from the blood. Such animals which may happen to be used as donors in blood transfusions are likely to transmit a serious form of babesiasis to the recipient.

#### TREATMENT

Nuttall and Hadwen have demonstrated, by means of carefully controlled experiments, the specific action of trypan blue for *B. canis*. The drug is used extensively in South Africa for the treatment of bovine and canine babesiasis. For the dog, a 1 to 2 per cent fresh, sterile, aqueous solution of trypan blue is used. In order to facilitate solution of the dye, Henning<sup>13</sup> recommends that it first be dissolved in a few drops of 90 per cent alcohol and then the required amount of boiled distilled water is added. The solution is injected intravenously under aseptic conditions, using from 5- to 25-cc amounts, depending upon the size and condition of the patient. In order to avoid shock, the solution of trypan blue preferably is administered slowly and at body temperature. The treatment is repeated within 24 hours if the desired result has not been obtained. The mucous membranes and skin take on a distinct blue color after treatment. This gradually disappears as the animal improves. This procedure has given good results in cases coming under our observations.

In addition to the intravenous injection of trypan blue, it would seem advisable to employ the use of stimulating tonics, especially those calculated to aid in regaining the normal hemoglobin content of the depleted blood. Careful nursing, very little exercise and nutritious foods such as milk, ground meat and eggs will aid the convalescent animal. Dogs treated by means of trypan blue continue to harbor the parasites in their system and are carriers of the infection the same as if having recovered without treatment.<sup>13</sup> Their immunity to a superim-

posed infection is said to be dependent upon the continued presence of the microparasite in their system.

Intravenous injections of trypaflavine (gonacrine) are reported as possessing a remarkable specific therapeutic action on *Babesia* and related infections.<sup>14</sup> By reason of its polyvalence, it has been suggested to replace trypan blue in the pharmacopeia. The dose for carnivora, as calculated by Dikmans<sup>15</sup> from that recommended by foreign workers, is 0.3 cc of a 2 per cent solution per pound of body weight. This drug has not been available in North America, but probably will be within the near future. According to a later article,<sup>16</sup> the subcutaneous or intramuscular injection of a preparation known as Acaprine (Bayer) is superior to either of the above-mentioned drugs in that only one dose is required and the tissues of the animal being treated are not stained.

#### SUMMARY

1. A chronic form of babesiasis due to *B. canis* has been encountered in various breeds of dogs in widely separated locations in Florida.

2. The training and coursing activities of affected greyhounds are seriously interrupted.

3. The brown dog tick, *R. sanguineus*, is suspected as being the principal vector of the infection.

4. The presence of chronic canine babesiasis as encountered was determined by blood inoculations and splenectomies of test animals.

5. Young pups splenectomized during or shortly following clinical reactions usually succumb within 24 to 48 hours as a result of severe relapse associated with numerous *B. canis* in the blood.

6. Pups in a state of relative immunity or premunized to *B. canis* did not react to intraperitoneal injections of blood from dogs showing symptoms of malignant jaundice, while non-immune pups reacted to such injections showing *B. canis* in the blood.

#### ACKNOWLEDGMENTS

Dr. J. H. Yarborough, of Miami, Florida, suspected the presence of canine babesiasis in the early cases coming under his observations and obtained favorable results by the use of trypan blue before the causal organism was demonstrated in Gainesville. The author is indebted to Doctor F. C. Bishopp, Bureau of Entomology, United States Department of Agricul-

ture, for identifying tick specimens and for giving the distribution of *R. sanguineus* and *D. andersoni*, and to Drs. John R. Wells, West Palm Beach; C. A. Palmer, Gainesville; H. C. Nichols and E. F. Thomas, Ocala, for aid given in obtaining strains of *Babesia*.

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## DISCUSSION

DR. JOHN R. WELLS: We should be deeply appreciative of Dr. Yarborough, whose keen imagination suspected this disease, and of Dr. Sanders, whose deft hands and quick eye discovered it and its traits.

This work is so new that I can add nothing to the splendid paper already given, but I should like to emphasize a few points made by Dr. Sanders with a view to their clinical application. To do this, I should like to give you two case histories and compare the findings with excerpts from the material presented in this paper. The first was a Boston terrier male about two years old. He came in with a history of depression. He had been shivering; he refused to eat; his membranes were slightly injected; he had a temperature of 104. I had recently vaccinated this dog, so I felt fairly sure he was not coming down with distemper.

About this time, we had been talking about piroplasmiasis, so just as a matter of course I made a blood-smear and had it examined in the laboratory. There were revealed just such beautiful specimens as you saw projected on the screen. In the meantime, I had given this dog a dose of castor oil. Later in the day, he cleaned out well. Dur-

ing the night, he ate his supper. The next day, he was normal in every respect. The blood has been negative ever since.

The paper states that "symptoms manifest by greyhounds and other breeds under observation were often somewhat vague, but in typical untreated cases, which often extended over a period of weeks, there occurred varying degrees of weakness, inappetance, vomiting, anemia, pallor of the mucous membranes, slight icterus of the sclera, intermittent fever, mucopurulent eye discharge, enlargement of the spleen and, less often, a cough." There you have all the symptoms of distemper and more, but this dog did not have primary distemper. I neglected to tell you this dog was clean; there were no parasites of any kind on him.

The paper states further that "on the sixth day following the initial reaction, it was difficult to demonstrate the parasites in carefully prepared blood-films of this test pup, piroplasms of doubtful identity being observed only rarely and by careful search." In the Boston we are considering, the blood was negative on the next day. In all artificially infected greyhounds an occasional parasite was observed, and the pup, in the meantime, recovered. The Boston made a spontaneous recovery.

Dr. Sanders goes on to say that "it should be remembered, however, that the ticks which transmit the disease while engorging may detach and drop from the animal several days before clinical symptoms of piroplasmosis are manifest." In the Boston, there were no ticks present.

About three years ago, I was called to see a mature setter that belonged to a physician. He had a high fever; he was depressed; there was some redness of the mucous membranes, and he would not eat. He had not been treated for distemper, so we made a tentative diagnosis of distemper. We gave him large and frequent doses of homologous serum, but he did not respond satisfactorily. Before the fifth day, we decided it might be a hemolytic streptococcus infection because his membranes had begun to blanch out. We sent some blood to the laboratory; it was examined on slides; it was cultured, and all results were negative. This dog became critically ill. He ran a fever as high as 107 at times. We gave him good nursing, frequent blood transfusions, saline infusions, and at the end of about six weeks he recovered by crisis.

About two years later, the same people bought a Doberman pup. He ran in the yard with the setters. The yard, by the way, was a tick-infested yard. One day he came down with very similar symptoms, with the exception that they were complicated by gastro-intestinal disturbances. We proceeded along the same line. After a few days, we had his blood cultured and examined thoroughly and the results were negative. The donor of these blood transfusions was the hospital donor. After frequent blood transfusions, the pup seemed to maintain more or less of a level condition; he got neither better nor worse. So the doctor and I decided, if this was a hemolytic streptococcus condition, the old dog which had had this thing not quite two years before would probably be carrying some bodies. So we took the blood from the setter and injected it into the Doberman pup and in 24 hours he was dead.

Undoubtedly, these were cases of piroplasmosis, and I bring this to your attention for this reason: If this disease finds its way into your locality, it would certainly be advisable to run periodic tests on your donor dogs so that you will not give piroplasmosis to some of the dogs you are trying your best to save.

Let me close with this word of admonition: Keep piroplasmosis in mind and follow its investigational progress. Remember that not many years ago, when the first reports of filariasis began to appear, you

probably felt secure in the belief that it was purely a tropical disease, but suddenly it appeared on your doorstep, so to speak, and instantly became your problem as well as ours. Forewarned is forearmed, so by way of anticipation, be prepared to cope with this disease should it appear in your locality, and thus maintain the high grade of professional service that your clients have come to expect.

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### Horse and Mule Association of America

A. B. Hancock, of Paris, Ky., noted breeder of race horses, was elected as president; Harry Stamp, of Roachdale, Ind., a breeder of pure-bred draft horses, first vice-president, and Louis E. Stoddard, of New York City, whose interests are chiefly in polo mounts, second vice-president, at the annual meeting of the Horse and Mule Association of America, held in Chicago, on December 2, 1936. Thus does the association continue its policy of supporting all breeds and types of horses and mules, without partiality.

F. M. Holmes, of New Britain, Conn., was elected treasurer, and Wayne Dinsmore, of Chicago, continues as executive secretary. Louis E. Stoddard, of New York; Ellis T. Early, of Cincinnati, Ohio; Frost Sparks, of National Stock Yards, East Saint Louis, Ill., and R. U. Carr, of Buffalo, N. Y., were the new directors elected.

The association is carrying on a national educational campaign to encourage the breeding, raising and use of horses and mules. This work, continued consistently for the past 17 years, has borne fruit in the production of more and better work and pleasure animals, and in a clearer understanding of their economic importance. National advertising in leading agricultural papers, to amplify the work of the association, is being continued.

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### Selenium Poisoning of Sheep

A heavy loss of sheep occurred in a gulch southwest of Pueblo, Colorado, after a herd had grazed there for a single night. The plants growing there were species of milk vetch, woody aster and lambsquarters. The U. S. Bureau of Chemistry and Soils has found that selenium, an element akin to sulfur, is poisonous to live stock when absorbed from the soil by plants.

The soil from that gulch contained three to five parts per million of selenium, while the milk vetch had 670 parts per million, the woody aster had 1,750 parts per million, and the lambsquarters had 890 parts per million. It is reported that any plant which contains over ten parts per million of selenium is dangerous for livestock to eat.

## THE PATHOGENESIS OF KETOSIS: PREGNANCY DISEASE OF SHEEP\*

By LEE M. RODERICK, G. S. HARSHFIELD and M. C. HAWN

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The problem of ketosis in sheep has received much study and yet certain features of its final etiology have remained obscure. Recent studies at this laboratory, therefore, have sought the ultimate explanation of the condition, for investigations had reached the stage where it had become a problem in experimental pathology rather more than of clinical medicine.

Reference<sup>1</sup> was made to losses among ewes which were well fed and under-exercised and which could not be explained by a sudden interruption of the feed or an insufficient nutritional intake. Among the recent writers on the subject, Belschner<sup>2</sup> seems to have found cases under similar circumstances. That situation is perhaps best explained on the basis of autointoxication and overloading of the maternal mechanism. Since the liver and kidneys must handle the metabolites of both ewe and the lambs, injury may very well occur. The condition occurs primarily during that period when there is a rapid growth of the lambs and their nutritional demands are consequently increasing. It would be difficult to prove experimentally that the products of fetal metabolism are in reality injurious for the ewe. We have not found fatty degeneration in the fetal livers removed from the affected ewes. No doubt some normal-appearing ewes in well fed flocks develop a moderate transitory ketosis, that is seldom recognized, unless the flock is regularly examined. This was a finding in the 1934-35 experiment flock. From a reading of Fincher's article,<sup>3</sup> it would likewise seem that ketosis is a far more common complication of bovine metabolism than has heretofore been suspected.

There may be distinct differences in the pathogenesis of ketosis in cattle and sheep from the standpoint of analogy, and yet Fincher<sup>3</sup> notes that ketosis is a disease of well nourished, high-producing cows. The livers and kidneys of two cases of bovine acetonuria, provided through the courtesy of Drs. L. Van Es and L. V. Skidmore, of the University of Nebraska, were examined and the fatty changes found to be similar to those in affected

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ewes. The pathology of ketosis in various animal species, including man, seems to be of the same character.

The relationship between the biochemistry and the pathology of the disease was considered in the previous publications<sup>1, 4</sup> from this laboratory. Sampson and his associates<sup>5</sup> have presented an excellent review of the fundamental biochemistry of ketosis, and Sampson and Hayden<sup>6</sup> have analyzed the relation of carbohydrates and ketosis. The observations at this laboratory agree with their findings, for reference<sup>1</sup> was made to the significance of the low blood sugar in cases of pregnancy disease and the liver glycogen was found<sup>4</sup> to be practically exhausted in clinical cases of the disease. The inability, however, to follow the abnormal physiology and pathology through its complete course in field cases has compelled us to resort to experimentally induced cases of the malady.

The pathological processes are already well advanced in the ewes when the clinical symptoms are recognized. Clinical and biochemic studies were useful in analyzing the etiological relationships, but such work could not be accepted as final proof until those theories were demonstrated experimentally. Furthermore, the usual failure of the therapeutic methods with sheep, which are successful in promoting recovery of ketosis in the human and in the bovine, still left a gap in the evidence. Leslie<sup>7</sup> did report 48 recoveries in 81 cases treated with glucose injections, while calcium gave no response.

The term "pregnancy disease" may be a misnomer, for while it is primarily a ketosis, it does emphasize the fact that pregnancy is an added metabolic load. The occasional case which comes to a prompt parturition sometimes recovers. It seemed logical therefore, that if the uterus could be evacuated, such procedures might be substituted for parturition. It has been impossible so far to evacuate the uterus of the ewes either with ecbolics or by mechanical manipulation. Careful hysterectomies and cesarean operations have been performed but the ewes died, all of which merely illustrates the fact that the sheep is an exceedingly unsatisfactory subject for experimental surgery. It would have been of no practical value in handling the farm flock if it had succeeded, but it might have contributed to a better understanding of the problem.

#### THE RELATION OF INADEQUATE NUTRITION TO THE FATTY LIVER AND KETOSIS

The recent papers mention the frequent occurrence of pregnancy disease in flocks which are fed on inadequate rations.

Reference<sup>1</sup> was made to the inferior quality of the feed, which was fed on many of the farms where losses have occurred. Such feeds are low in digestible carbohydrate and high in cellulose. Similar observations were made by Elder and Uren,<sup>8</sup> Rose,<sup>9</sup> and Dayus and Weighton.<sup>10</sup>

Many of the affected sheep are in reality thin and in poor condition, but some comments by Leslie<sup>7</sup> are particularly pertinent. It is invariably associated in his experience with loss of body weight, undernutrition and with unbalanced and deficient feed. The inaccessibility of feed during bad weather was often followed by outbreaks. Two serious losses followed 24-hour railway journeys. Hopkirk<sup>11</sup> likewise reports that losses were produced in sheep, which were fed so that they lost weight, even though they were given a full mineral mixture. The question now arises how these factors of partial starvation and hunger produce fatty changes in the liver and subsequent ketosis.

There are very little data available on the effects of fasting on the ovine metabolism. Benedict and Ritzmann<sup>12</sup> concluded that while man develops acidosis during fasting, herbivora do not, for they excrete very small amounts of acetone bodies. They did mention a rather significant observation by Palladin, that acid formation may follow starvation in sheep. Sjollema's earlier work is cited to show that the cow does not easily produce much acetone except in certain diseased conditions.

Sjollema<sup>13</sup> states in a more recent article, however, that in ketosis, the metabolism of carbohydrate is reduced to such a degree that the oxidation of fat cannot be carried beyond the stage of ketone bodies, that the liver is largely concerned in the metabolism of fat and that the ketone bodies are formed mainly or solely in that organ. Wester<sup>14</sup> notes that ketosis is essentially a problem of cattle and sheep and that acetonuria is induced in cows by withholding of feed for two to six days. Best<sup>15</sup> states that the liver is an important site for the formation of ketone bodies.

Schmidt and Wingen<sup>16</sup> write that a great need exists in the latter half of pregnancy, which cannot be met completely through nutrition and is covered by the glycogen depot in the liver. This whole process of acid-base equilibrium varies physiologically according to the degree of carbohydrate deficiency in the metabolism of pregnancy. While that was written for the human, it is likewise quite a *propos* for the sheep. The quotation by Bloor<sup>17</sup> furthermore seems appropriate:

When the liver is full of glycogen, fat cannot be deposited there in notable amounts, but when the glycogen is exhausted or low, fat flows in to take its place.

## THE CALCIUM THEORY OF CAUSATION

We quite agree with Dimock, Healy and Hull<sup>18</sup> that the prevention of this condition is primarily a question of proper nutrition and care of the ewes during pregnancy. We do not agree, however, that the increased demands on the maternal supply of calcium during the last two months of pregnancy explains why this disease develops so frequently in sheep. A careful and thorough search of the biochemic literature reveals little to suggest that the blood calcium is concerned in the acid-base equilibrium. In the metabolism of calcium, it is excreted to a large extent through the bowel rather than the urine.

Little mention can be found of any relation of calcium to fatty changes in the liver. Losses from pregnancy disease have been encountered among ewes fed on alfalfa hay, which is high in calcium. Fortunately, however, that is of rare occurrence. Some of the affected flocks under field conditions have had a mineral mixture and the disease was produced in the following experiments in ewes which have had a liberal mineral supplement in their grain ration. The intravenous injection of calcium solutions is of little value in treatment. This is the routine experience of various workers including this laboratory although an occasional report implies its efficiency. Sampson and Hayden<sup>6</sup> found normal blood-calcium levels in both ovine and bovine cases of ketosis.

## EXPERIMENT EWES

*Flock of 1934-35:* They were rugged, 5-year old, crossbred Hampshire-white face ewes. They were fed from December 20, 1934, on alfalfa hay *ad lib.* and a grain mixture of bran, oilmeal, oats, and barley. They received 1 pound daily at the start, but this was soon increased to 1½ pounds per ewe. They had 10 grams daily of a calcium, phosphorus, iodine mineral mixture with the grain. They secured little exercise and soon were in excellent condition. When four of the ewes were selected for experiment (February 20, 1935), two were found with a well developed acetonuria. They were in advanced pregnancy.

*Flock of 1935-36. Lot I:* The ewes were of the same stock as the previous year. They were fed on alfalfa hay *ad lib.* with from 1½ to 2 pounds of grain and 10 grams of a mineral mixture per ewe per day. The grain consisted largely of cracked corn with some ground oats and oil meal while the mineral mixture consisted of equal parts of ground limestone and bonemeal. They had little exercise and the average gain was 39 pounds in 87 days. The urine of these ewes was tested twice a week for a

month for acetone and its pH, but no abnormalities were found. About three weeks before term, the following trials were made. Three of the heavier ewes were confined to a box stall but with liberal feed. Ketosis appeared in one on the fifth day and persisted until the ewe was turned out in the yard when the condition subsided. The five other ewes were changed to brome grass hay and a minimum quantity of oats plus the mineral mixture. Ketosis appeared in 24 hours in one of the ewes and continued until the previous liberal ration was restored.

*Lot II:* Another group of eight ewes were fed on corn stover with some brome grass hay. They were fed merely enough ground oats, about one-half pound daily, to enable them to consume the mineral mixture. The gain for the entire group was only 67 pounds in 87 days. The urine was checked twice a week. One ewe developed ketosis but it disappeared when the liberal feed of lot I was provided.

#### STARVATION EXPERIMENTS

*Experiment I:* Four ewes from the 1934-35 flock were placed in bare box stalls, February 12, 1935. They had water but no feed. An acetonuria and acidosis developed in two of the ewes within two days. Ewes 257 and 266 received no further feed and were destroyed with the characteristic chemical and pathologic features of pregnancy disease. A biopsy was performed on ewe 261 on the third day of the fast and a section of the liver removed. It was loaded with fat, with scarcely a normal liver cell to be seen. The ewe recovered with dextrose injections, reared twin lambs and when slaughtered (March 12, 1936) was found to have quite normal appearing liver and kidneys. Ewe 263 was fasted until February 15, when dextrose was given and the feed restored, which was followed by recovery.

TABLE I—*Experiment I. Urine examinations.*

EWE	DATES													
	2-14		2-15		2-16		2-18		2-19		2-21		2-25	
	A	pH	A	pH	A	pH	A	pH	A	pH	A	pH	A	pH
257	Tr	7.2	+	6.4	+	6.4	+	6.8	+	6.8	+	5.8		
261	+	6.2	+	6.2	Tr	7.0	+	7.0	+	6.2	+	6.2	N	7.0
263	+	7.2	+	6.6	N	8.2	N	8.4	N	7.8	N	7.8	N	7.4
266	Tr	6.6	N	6.4	+	6.4	+	6.4	+	6.4	+	6.2	+	6.4

*Key*

A = Qualitative test for acetone.

N = Negative.

+

*Experiment II:* Four ewes were withdrawn from the 1934-35 flock on February 20, 1935, and placed in the barn on their usual feed for preliminary observation. Two of them already presented a ketosis on a good ration plus the mineral mixture. Starvation was started in bare box stalls on February 25 and the ewes handled as noted in Table II. A biopsy was done on the livers of ewes 258 and 264, while still in the early stages of ketosis. They died, even though they arose and ate within one-half hour after the operation. Ewe 262 was slaughtered for fat determination on the liver. Ewe 265 promptly improved when the feed was restored on February 28.

TABLE II—*Experiment II. Urine examinations.*

EWE	DATES															
	2-20		2-22		2-25		2-26		2-27		2-28		3-1		3-4	
	A	pH	A	pH	A	pH	A	pH	A	pH	A	pH	A	pH	A	pH
258	+	7.8	+	7.8	+	8.0	+	6.2	+	6.8						
262	N	7.8	N	7.8	N	7.8	N	6.8	Tr	6.8	+	6.4	+	6.2		
264	+	8.0	N	7.8	+	8.0	+	5.8	+	6.0	+	7.4				
265	N	7.8	N	7.6	N	7.8	Tr	6.4	+	6.2	+	5.6	+	7.4	N	8.0

*Key*

A = Qualitative test for acetone.

N = Negative.

+ = Positive.

Tr = Trace.

We were able, therefore, to reproduce all of the usual pathologic processes both morphologic and chemical, which we regularly recognize in clinical pregnancy disease, in well fed ewes, by the simple expedient of starvation. The deposition of fat in the livers is a progressive condition, for it is nominal in early stages of ketosis and attains enormous proportions with continued fasting. The amount of fat in the liver and the period of ketosis was rather closely correlated with the duration of hunger. The changes in the livers and kidneys of these experiment ewes seemed identical with those found in field cases of pregnancy disease.

*Experiment III. Lambs:* The next step was to try to eliminate the element of pregnancy and to show indirectly that it is likewise an increased metabolic load and drain on the ewe. Three lambs weighing from 78-90 lbs. were starved for 35 days although water was freely supplied. They remained bright and alert until they were slaughtered. The losses in weight were, respectively, 15, 14 and 28 pounds. One error was committed in permitting

a thin layer of litter to remain in the stall, for the lambs proceeded to lick the very floor and the onset of ketosis was delayed. Acetonuria did not appear for 35, 19 and 31 days, respectively. The pH of the urine never reached the acid range, although ammonia was appearing at the end of the experiment. Hunger, however, had produced a marked deposition of fat in the livers.

*Experiment IV:* In the next trial three lambs were confined to a bare scrubbed concrete stall. It was interesting that lamb 270 ground its teeth similar to the field cases of the disease. (Table III.)

TABLE III—Data from experiment IV.

LAMB	INITIAL WEIGHT (LBS.)	LOSS OF WEIGHT IN 8 DAYS (LBS.)	DAYS REQUIRED TO PRODUCE QUALITATIVE TEST FOR ACETONURIA
50	101	17	10
175	97	18	7
270	93	11	3

The pH of the urine remained alkaline through the experiment. In another trial with two lambs, weighing about 65 lbs., we were able to acidify the urine through starvation in four to five days, although it took 10 and 11 days, respectively, to produce acetonuria. While one lamb in the above experiment developed mild acetonuria by fasting for 3 days, four others required 7 to 11 days. No pregnant ewe in these trials has been able to stand that period of starvation, for among eight ewes, two were showing ketonuria even while on good feed and the six others manifested it within 48 hours.

*Experiment V:* Three lambs were fed on an exclusive ration of wheat straw, with no grain for a period of two months. The reaction of the urine was checked twice a week, but it remained alkaline, nor was acetonuria detected by the qualitative method. The lambs appeared bright and alert and yet when they were slaughtered and a chemical analysis made of the livers for fat, it was found that the changes were advanced. The results of the chemical examination are given in table IV.

The histological examinations of the livers of the lambs were directly correlated to the above chemical results. The liver of lamb 24 presented an extreme degree of fatty change, for not a single normal liver cell was detected. Fat was likewise appearing

in the kidneys. The stain showed the least fat in the liver and kidney of lamb 3.

It seems highly probable that ewes would have developed ketosis in a correspondingly shorter period. Four ewes were shifted from a daily ration of alfalfa hay with  $1\frac{1}{2}$  lbs. of grain to  $\frac{3}{4}$  lbs. of grain with wheat straw. One ewe developed ketosis in 5 days. The others remained normal for 3 to 4 weeks. Reference was made in Bulletin 261, p. 11, to a serious outbreak of ketosis which followed the substitution of sweet clover straw for alfalfa.

TABLE IV—*Chemical examination of livers for fat.*

SHEEP	CRUDE FAT	
	FRESH BASIS	DRY BASIS
3	9.12	23.39
27	14.79	40.86
24	20.64	50.79

Fatty livers have accordingly been produced by the process of starvation in a series of twelve ewes and lambs, in which the amount of fat has been determined. The amount of crude fat in the fresh liver varied from 9.12 per cent to 34.75 per cent. The amount is directly correlated to the degree of inanition and the period of starvation. The amounts of fat in these experiment sheep corresponded to those found<sup>4</sup> in the series of field cases of pregnancy disease.

#### DISCUSSION

It seems, therefore, that this problem is quite definitely related to carbohydrate metabolism. With an inadequate carbohydrate intake, the glycogen is withdrawn from the liver to maintain the blood-sugar level in metabolism, and fat takes its place. While the increased excretory burden associated with pregnancy may provoke some liver injury, the fatty changes in the liver and kidney are readily produced by starvation and an inadequate nutritional intake. There are unpublished experimental data to show that the new born lamb is provided with a reserve of glycogen in its liver. The fetal lamb liver is storing glycogen for some days prior to parturition, providing the ewe is adequately fed. As high as 9 per cent of glycogen has been found in the livers of these lambs, which is two or three times as much as in the liver of the ewe. The demands of a twin pregnancy are thus apparent.

Biopsies have been done and portions of the liver removed surgically for histological examination on three ewes and five lambs which showed evidence of ketosis from starvation. Biopsies were tried on field cases of pregnancy disease but they were too far gone for such experiments. It was necessary to use sheep in which the fatty deposition had been induced. Three of the lambs and ewe 261 survived and were fed until they were quite normal, when they were slaughtered and examined. It was found that, when these sheep recovered and the ketosis disappeared, the fat was gradually removed from the liver. This demonstrated the reversibility of the reaction between the presence of glycogen and fat in the livers of sheep.

This experimental work demonstrates the part that semi-starvation and inadequate feed play in the causation of the disease. The nutrition of ewes should be such that their condition improves with advancing pregnancy. The animal body has a greater reserve of fat and protein than of carbohydrate. The use of a liberal and balanced ration is needed, yet the use of molasses to supplement roughages of low quality seems justified on fundamental principles.

#### ACKNOWLEDGMENT

We have had the helpful coöperation of T. H. Hooper and A. J. Pinckney, of the Department of Agricultural Chemistry, in some of the analytical work.

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#### DISCUSSION

DR. C. F. SCHLOTTHAUER: I am very much interested in the discussion on ketosis because I have done some work on this disease in sheep. I was interested especially in the thought that the fatty liver preceded the ketosis. In our work with it, we were of the opinion that it was somewhat reversed. Sheep suffering from ketosis stop eating, and

fasting will cause deposition of fat in the liver. We found fatty livers in all the sheep on which we conducted experiments.

We did find, however, that regardless of what the sheep were being fed, if we increased the exercise of the flock this disease did not develop. A ewe which is pregnant with twins exercises less than the one which has one lamb; and as they become heavy with lambs, they exercise less and less, unless you force-exercise them. I am of the opinion that this accounts for the increased incidence of this disease in ewes with twin pregnancy or more.

For force-exercising sheep, we place the feed at least a quarter of a mile from the barnyard and force them to walk out to it. In the winter time, we have to plow a path through the snow for them. By following that routine, we have not had any trouble. However, if any of the farms fail to follow this routine, we do have trouble.

For several years this system has worked very well. However, we had so much snow last winter that it became too deep to plow a path through it. As a result, the force-exercise system did not function and we had a lot of trouble. Sheep will not exercise unless you make them. If they can stand still and eat, they will do it, and that is especially true of ewes pregnant with twin lambs or triplets.

From our work, we are of the opinion that fatty liver is secondary.

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## CULTIVATION OF THE VIRUS OF INFECTIOUS BRONCHITIS\*

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In January, 1935, a respiratory disease of young chicks was encountered in a broiler plant that had a total population of about 40,000 of all ages. Day-old chicks were added every week and each lot contracted the disease when it was about nine days old. Although the mortality was of some consequence, poor growth of the survivors constituted a greater loss.

The symptoms of the disease were similar to those of laryngotracheitis, and the owner thought that vaccination at a young age might solve the problem. But, when young chicks were vaccinated and then exposed in the laboratory to natural cases from the plant, they took the disease in spite of having shown "takes" following vaccination. Moreover, on autopsy the bronchi contained mucus or caseous plugs and the air-sacs were either turbid or contained caseous material. Fibrinous exudate occasionally was seen in the pericardial sac. From these observations it was evident that we were not dealing with laryngotracheitis.

A review of the literature showed that this disease was undoubtedly the same as that reported by Schalk and Hawn,<sup>1</sup> in 1931, and by Bushnell and Brandly,<sup>2</sup> in 1933, although the latter investigators referred to it as laryngotracheitis. Beach and Schalm<sup>3-5</sup> appear to have seen the same infection in California.

On February 28, four chicks were received from another farm and showed the lung and air-sac lesions mentioned above. Material from these was emulsified and 1.0 cc inoculated intratracheally into each of two birds that had been vaccinated against laryngotracheitis on February 11. By the fourth day, there was a profuse nasal discharge and the birds were killed on February 5. There was a rhinitis and laryngitis, caseous plugs in the lungs and exudate in the air-sacs. The exudate was collected, ground in broth with sterile sand and centrifugalized. The supernatant fluid was passed through each of two Berkefeld V filters and the filtrates inoculated intratracheally and intranasally into each of two birds in amounts of 1.5 cc. One of the filtrates was contaminated but the respiratory symptoms of this bird on

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the following day (March 6) were not so pronounced as those exhibited by the bird that received the sterile filtrate.

A third bird received the same dose of non-filtered material and showed a hacking cough on the following day. The respiratory symptoms gave way to a nasal discharge on March 11. A fourth bird was inoculated intracloacally and showed slight reddening of the membrane on the following day. This was definitely inflamed for the next two days and disappeared by March 11, but on this day a nasal discharge made its appearance. A non-inoculated control kept in the same room exhibited the first mild symptoms on March 11 and these were more pronounced the following day. On March 12, the five birds were vaccinated against laryngotracheitis and all but the control showed definite "takes" within 48 hours. The control was revaccinated on March 15 and then showed a definite "take." The nasal discharge had cleared up in all birds except the control by March 14.

On March 12, some of the nasal discharge was collected from the above birds and combined in suspension with autopsy material from four more chicks from the above outbreak and frozen overnight. On the following morning, part of the suspension was filtered through Berkefeld V, N and W filters and a Seitz disc. The filtrates were inoculated in doses of 1.5 cc intratracheally and intranasally. Sterility tests showed, however, that the V filtrate was contaminated. A fifth bird received the same dose of non-filtered material and a sixth was held as a control. Table I shows the observations made on these birds.

From table I it is evident that the virus passes Berkefeld filters. In this case the Seitz disc seems to have retained the virus,

TABLE I—Results of inoculation of filtered material on March 13.

BIRD	INOCULUM	CLINICAL OBSERVATIONS ON:				
		3-14	3-15	3-16	3-17	3-18
1	Berkefeld V (1 min.)	Severe respiratory symptoms, 4 P. M.	T. 42.6	T. 42.1.	Occasional gasping	T. 42.8
2	Berkefeld N (40 sec.)	Mild respiratory symptoms, 4 P. M.	T. 42.8	T. 42.5.		
3	Berkefeld W (1 min.)	Very slight respiratory symptoms, 4 P. M.	T. 42.5.	T. 42.1.		
4	Seitz (2 min.)	Normal	T. 42.2	T. 42.2	Occasional gasping	T. 42.8
5	Non-filtered	Marked rales	T. 42.6.	T. 42.0.		
6	Control	Normal	N. D.	N. D.		
			T. 42.0	T. 42.0		T. 42.8

T. = temperature.

N. D. = nasal discharge.

although the bird inoculated with this filtrate, as well as the control, had contracted the infection by March 18. This strain of virus was carried for several months by bird passage but was finally lost.

From the time the first outbreak was encountered, the disease has been seen on many occasions. Usually it occurs in young chicks, but occasionally the disease is spread to the adults on the same farm in those cases where the infection has been brought in for the first time.

#### PROPAGATION IN EGGS

Material from an outbreak of the disease was filtered through each of two Berkefeld N filters on April 7. Both filtrates were sterile. One was inoculated in doses of about 0.4 cc into the chorioallantoic membrane of each of four twelve-day-old eggs and the other into three eggs of the same age. Each filtrate was held in freezing chamber for two days and inoculated into experiment birds intratracheally. Both birds developed respiratory symptoms within 48 hours. Eleven days later, each bird was inoculated intracloacally with laryngotracheitis virus and each showed marked inflammation within 48 hours.

The embryos of two of the four eggs inoculated with one filtrate were dead the following day, but the other two were still active when the eggs were opened six days after inoculation. In neither case did the membranes show any of the changes that are seen in eggs infected with the viruses of laryngotracheitis, fowl-pox or pigeon-pox. One membrane was slightly thicker than the other and was used as inoculum for the second generation.

Of the three eggs inoculated with the second filtrate, two embryos died on the second and fourth days, respectively, and the third was still active when the egg was opened on the sixth day after inoculation. Again there was no gross evidence of infection. However, when an emulsion of the membrane used to initiate the second generation was inoculated intratracheally on the day it was harvested (April 13), it provoked the disease in less than 48 hours. Seven days later, the bird was inoculated intracloacally with egg-propagated laryngotracheitis virus and showed a "take" in the usual time.

The technic employed in subsequent passages was as follows: The membrane was harvested from the egg under strictly aseptic conditions. A platinum loop was passed over a large portion of the membrane as it lay in a Petri dish and then streaked on an agar plate as a sterility test. If reinoculation was to be made at once, the membrane was ground in a sterile mortar with about

5 cc of broth. The emulsion then was transferred to a sterile tube and centrifugalized, after which the supernatant fluid was drawn into a syringe fitted with a bent needle to facilitate inoculation. After the last egg of a series was inoculated, about 0.5 cc of the fluid in the syringe was injected onto an agar plate and streaked over the surface as a sterility test.

If no inoculations were to be made immediately, the harvested membranes were cultured and placed in suitably labeled sterile tubes and stored in the freezing chamber. In this way a constant sterility check was made on every membrane and inoculum.

The inoculated eggs were candled daily up to the time of harvest. Eggs containing dead embryos were stored under refrigeration until the eggs of that generation were harvested.

Table II indicates briefly the subsequent generations, age of eggs at time of inoculation, age of virus, number of eggs inoculated and number discarded because of early death (dead on day after inoculation) and, finally, the number remaining with the individual records on these. The letter "A" means that the embryo was alive and the number following indicates the days

TABLE II—Showing the results of the propagation of bronchitis virus from the second to the fourteenth generation.

GENERATION	AGE OF EGGS AT INOCULATION (DAYS)	AGE OF VIRUS (DAYS)	EGGS INOCULATED	EGGS DISCARDED BECAUSE OF EARLY DEATH	EGGS REMAINING	RECORDS OF INDIVIDUAL EGGS*
2	11	0	4	0	4	A7, A7, A7, A7
3	11	0	3	1	2	A7, D3
	9	0	2	1	1	A7
4	11	1	4	1	3	A6, A6, A6
5	10	0	3	0	3	A7, A7, A7
6	12	0	1	0	1	A5
	11	0	2	0	2	D4, A6
7	8	2	3	0	3	D6, D5, D4
	11	2	3	0	3	D2, D4, D6
8	11	1	5	0	5	D6, D6, A6, A6, D6
9	12	1	4	0	4	A6, D6, A6, A6
10	13	0	5	1	4	D4, D4, D4, A4
11	10	0	6	2	4	D4, D4, D4, D4
12	10	2	5	1	4	D6, A6, A6, D6
13	11	8	4	0	4	D4, D4, A4, D4
14	12	0	5	0	5	D4, D3, D3, D4, D4

\*A7 = alive on seventh day; D3 = dead on third day, etc.

of incubation. The letter "D" means that the embryo died and the number indicates the day of death after inoculation.

#### EFFECT ON EGGS

As far as we are able to observe, death of the embryo is the best evidence of growth of the virus. It will be seen from table II that in the first six generations very few embryos died, even though the eggs were incubated from five to seven days after inoculation. However, from the seventh generation on, the majority of embryos died in a relatively short time.

Death cannot be attributed to bacterial action because not a single egg was found to be infected. The size of the dose was not responsible for death, because in our experience eggs regularly tolerate doses of 0.5 cc and only a few young embryos are affected by a dose of 0.8 cc. As further evidence that mere size of dose was not operative, it may be cited that during these experiments eggs of the same age were inoculated regularly with the viruses of laryngotracheitis, pigeon-pox and fowl-pox in doses as great or greater. In the case of eggs inoculated with the pox viruses the embryo usually lives up to the time of harvest (6 to 7 days) in spite of greatly thickened membranes. When laryngotracheitis virus is passed rapidly, it may be made to kill embryos quite regularly in three days. However, the virus can be inoculated in such a way as to permit the embryos to live five to seven days. Thus, in the same incubator we have had eggs inoculated with bronchitis virus die rather early, whereas eggs of the same age inoculated with the viruses of laryngotracheitis or with fowl-pox and pigeon-pox continue to live up to the time of harvest.

In no case have membranes shown the lesions found in membranes in which laryngotracheitis or the pox viruses are cultivated. Consequently one may utilize the lesions provoked by laryngotracheitis on the chorioallantoic membrane as a means of distinguishing this virus from that of bronchitis.

Very rarely a membrane may show two or three slightly turbid areas which were not considered to be the result of traumatism. This was seen in one egg of the third, seventh, 13th and 14th generations, respectively. The membranes appeared as thin or even thinner than those of non-inoculated eggs and they seemed to adhere to the shell more tenaciously. The blood-vessels seemed to be more injected than in non-inoculated eggs.

Usually the embryo appeared smaller and more shrunken than a normal embryo of the same age or an embryo from an egg in which other viruses had been propagated. Actual weights

were made on only one occasion. The ten eggs had been set on June 8, and on June 18, five were inoculated with fowl-pox virus, four with bronchitis virus and one was held as a control. All ten eggs were opened after seven days of incubation and weighings made of the embryo after removal of the membranes and yolk sac. The five pox embryos were alive and weighed 18, 13, 12, 17 and 17 gm, respectively, or an average of 15.4 gm. Two of the four bronchitis embryos (first and last) had died the previous day and the weights were 8, 13, 13 and 8 gm, respectively, or an average of 10.5 gm each. Obviously the two dead embryos were smaller than the two living ones. The live embryo from the control egg weighed 22 gm.

In a few eggs the yolk was quite solidified but there is no proof that this is associated with the activity of the virus. However, in most cases the residual albumin seemed to be very watery.

No histological studies have been made.

#### ANIMAL INOCULATIONS

All birds used in these experiments were grown in complete confinement in an isolated room in the poultry building and were brought to the animal rooms only when needed for experimentation.

It has been mentioned already that the membrane emulsion used to initiate the second generation caused a respiratory disease in an inoculated chicken which was afterwards shown to be susceptible to the virus of laryngotracheitis. This may have been merely a question of the survival of virus in the inoculated egg, but inoculations from eggs of subsequent generations continued to produce the disease, as will be described.

Four birds were vaccinated with egg-propagated laryngotracheitis virus on March 25 and were shown to be immune to this infection by intratracheal inoculation on April 11. On April 20, each bird was given 0.5 cc intratracheally of an emulsion of membranes of second-generation virus and within 24 hours pronounced râles were observed.

The emulsion of membranes of the egg used to initiate the tenth generation was used also to inoculate two half-grown birds intratracheally and intranasally in the afternoon of June 8. These birds ate very little the following day and by the next morning showed respiratory symptoms. Moist râles were still evident nine days after inoculation. Two other birds were inoculated subcutaneously at the same time but continued to eat. However, one of these developed the disease later, probably hav-

ing contracted it from the first two, although they were in separate cages. Fourteen days after inoculation, the four birds were tested and found to be susceptible to laryngotracheitis.

Material from two eggs of the 13th generation was emulsified and centrifugalized for the following inoculations:

Two birds immunized against laryngotracheitis on June 16 and shown to be immune on June 26 were inoculated with 0.8 and 1.0 cc, respectively, on July 7. The first bird showed mild râles on July 14 but never developed a nasal discharge. The second bird showed gasping symptoms and a nasal discharge on July 10. Reinoculation on July 16, with 14th generation virus, was without effect.

Two birds (immune to tracheitis) were each given 0.8 cc of the same bronchitis virus subcutaneously. One of these showed the first symptoms on July 16, the day each was reinoculated with 14th generation bronchitis virus. The bird that was not affected on July 16 showed râles three days later.

Two more birds (immune to tracheitis) were each given 0.8 cc of the same bronchitis virus intramuscularly on July 7 and each was reinoculated as above on July 16. One of these may have shown mild symptoms before the second inoculation but none were observed subsequently.

Two birds (immune to tracheitis) were inoculated intracloacally on July 7. Two days later, each showed a mild inflammation which increased the following day and subsided the next day (July 11), at which time one of the birds showed mild râles. Reinoculation with 14th generation virus on July 16 provoked no symptoms.

About this time, we received three birds from a spontaneous outbreak, thought to be bronchitis. On July 7, all had recovered and two of these were shown to be susceptible to laryngotracheitis and the third resisted an intratracheal inoculation of the 13th generation bronchitis virus used above. Finally, on July 14, this bird was inoculated with laryngotracheitis virus and died four days later.

Only one neutralization test was attempted. On July 16, each of two birds (immune to laryngotracheitis) received 0.5 cc intratracheally of a mixture of equal parts of broth and a 14th generation bronchitis virus emulsion. These birds developed typical symptoms on July 18. Two other laryngotracheitis-immune birds that received 0.5 cc each of a mixture of equal parts of the same virus suspension and serum of birds recovered from bronchitis remained unaffected.

The mixtures of broth and virus and serum and virus were held overnight at about 4° C. and inoculated in duplicate the following day, in the same dose as before, into birds that were not immune to laryngotracheitis. The birds that received serum-virus never showed symptoms. One that received broth-virus mixture showed symptoms on July 18. The status of the other bird was questionable but this bird was definitely infected on July 20.

A control bird (immune to tracheitis) was given 0.5 cc of the virus emulsion on July 16 and exhibited questionable symptoms, but by July 21 a nasal discharge was in evidence.

#### SUMMARY

1. The virus of infectious bronchitis passes all grades of Berkefeld filters.

2. Introduced intratracheally and intranasally the virus regularly provokes the disease. Subcutaneous and intramuscular inoculations are either non-infective or else produce the disease after a prolonged incubation period.

3. Inoculated into the chorio-allantoic membrane of the developing embryo, the virus does not produce gross lesions such as are produced by the viruses of laryngotracheitis, fowl-pox and pigeon-pox. The absence of such lesions could be used to distinguish between the viruses of laryngotracheitis and bronchitis.

4. After a few passages, the virus developed the capacity to kill the embryo. The embryo appears smaller than embryos of the same age from eggs in which pox and laryngotracheitis viruses have been cultivated.

5. After passage through 14 generations, the virus was infectious for chickens that were immune to laryngotracheitis. Likewise, birds recovered from bronchitis were still susceptible to laryngotracheitis.

6. The serum of birds recovered from bronchitis is capable of neutralizing the virus.

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#### DISCUSSION

DR. F. C. TUCKER: I would like to ask Dr. Beaudette if it would be possible for a man in the field to differentiate between bronchitis and laryngotracheitis.

DR. BEAUDETTE: Not definitely, although that would depend to some extent upon the age of birds that was affected. That is to say, if you were to encounter an outbreak of a respiratory disease in chicks three, four or five days old, which killed a large number of them, and if, on postmortem examination, you found thick mucus or cheesy plugs in the bronchi, and if you found a turbidity of the abdominal air-sacs, or a fibrinous exudate in the pericardial sac, I believe you would be justified in making a diagnosis of bronchitis.

On the other hand, if you were to meet with a mild respiratory disease in half-grown birds, and especially if it were to occur during warm weather, at which time laryngotracheitis is not always so fatal, then one should certainly hesitate to make a definite diagnosis. When birds which show the symptoms that I have just described are submitted to us for diagnosis, we simply place them in an animal-room, where they cannot contract any other infection and permit them to recover. Then if we have three or four birds, we will vaccinate a couple of them against laryngotracheitis and inoculate the other one or two with the virus of bronchitis, and await results.

DR. HUGH HURST: In the field, we find cases where chicks or pullets two to three months of age have a sort of dry, gasping, respiratory disturbance; there is no mucus, no water of any sort, no excretions, and those flocks very often clear up in a period of three or four days, with seldom any loss. Do you have a name for such a condition?

DR. BEAUDETTE: I am afraid I cannot answer your question definitely. It might be this infection, although I think if one observes an outbreak of this disease rather closely from the beginning, he will see rather pronounced respiratory symptoms for a day or two, and these will rapidly give way to a slight nasal discharge, which in turn clears up in about a week.

DR. HURST: This condition Dr. Beaudette has described is something we have in some flocks. We have others in which there are no excretions whatsoever, and it may be just a milder attack of the same condition. We do have a lot of those cases so typically depicted in his last statement, but there are many chicks which do not have any sort of discharge, and I have been unable to give people any satisfactory explanation of what it is.

DR. E. E. CLORE: I had a little experience this year that I think might be interesting. It happened in a brooder-room containing 14,000 baby chicks. In this brooder-room, we had an outbreak of bronchitis. These chickens were from one to 14 days of age. In our brooder-room, we had been doing some work in the crossing of breeds, particularly the crossing of the Leghorn hen and the Australorpe rooster, which gives us an Austra White. We found, after this bronchitis occurred in our brooder-room and we had lost about 8,000 of the 14,000 chicks, that the Austra Whites were not nearly so susceptible to bronchitis as the other birds. Now whether the crossing had anything to do with this, I do not know. They were all living under the same ideal conditions, in the same room, but nevertheless we did not have as much trouble in the cross-bred chickens as we did in the pure-bred.

DR. C. A. BRANDLY: I think this is the first report of the egg propagation of so-called bronchitis virus. I think there is no question as to its importance, in view of the very great prevalence of this condition as it has occurred for several years. We have noted it particularly in the states of Illinois and Missouri during the past hatching season; and inasmuch as the condition is so important, and since it is sometimes confused with coryza—of which we are told there are numerous types and causes—egg propagation as a probable aid in diagnosis is indeed significant. I think Dr. Beaudette deserves considerable credit for the investigation he has carried on with this problem.

DR. CLORE: I would like to ask Dr. Beaudette if he knows of any vaccination method in use on these baby chicks? I have heard some rumors of it. This disease is one which is very important to the hatchery men, and it is something they have been unable to cope with up to this point, except by means of sanitation.

DR. BEAUDETTE: Soon after we realized that we were dealing with a different infection from laryngotracheitis, we inoculated some half-grown birds intracloacally with this virus and produced an inflammation; and later, we found that the birds were immune to the infection.

In this paper, I believe I mentioned inoculation of a bird intracloacally, but that bird came down with the disease in four or five days—perhaps it was a shorter time—after the intracloacal inoculation. Consequently I do not know whether the intracloacal inoculation in that case infected the bird in the respiratory tract, or whether this particular bird contracted the infection from some other bird in the animal-room, which was inoculated at the same time in the respiratory tract.

Personally, I was not interested in attempting to immunize against the infection for the reason that chicks appear to be too small to immunize, and any method of immunization would have to be applied after the age of six weeks, which would be too late because at that age the disease is of little consequence.

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### Serum Marketing Agreement

An order regulating the handling of anti-hog cholera serum and hog cholera virus was signed December 2, 1936, by Secretary of Agriculture Henry A. Wallace. The order makes effective a marketing agreement developed early in 1936 by officials and technical experts of the Department of Agriculture in consultation with representatives of the serum industry.

The order and agreement, which became effective December 7, 1936, are intended to insure the maintenance of an adequate supply of anti-hog cholera serum and hog cholera virus for any emergency and to aid in making certain improvements in trade conditions. The plan is authorized by an act of Congress which established, as a national policy, this form of aid in combating hog cholera.

The law authorizes the Secretary of Agriculture to consult with manufacturers of serum and virus with respect to commercial handling and distribution of such products that move in interstate trade or foreign commerce.

The agreement is to be administered partly through a control agency, representing the industry, that will deal especially with commercial problems. The Division of Virus-Serum Control of the U. S. Bureau of Animal Industry will deal with the scientific and veterinary features of the agreement.

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The evil that men do lives after them. The good is oft interred with their bones.—Shakespeare.

## RESULTS OF ADMINISTERING TWO CALFHOOD INJECTIONS OF A LIVING BRUCELLA ABORTUS CULTURE TO PREVENT BANG'S DISEASE\*

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Observations on heifers injected during calfhood with living *Brucella abortus* cultures to prevent Bang's disease suggested the possibility that greater resistance may be enhanced by the administration of more than one injection. This paper summarizes the results of an experiment with eleven heifer calves. Eight calves were injected twice, and three calves were kept as controls. All calves were negative to the agglutination test before they were injected.

A strain was selected for the calfhood injections which had been under artificial cultivation for two years, and possessed a moderate degree of pathogenicity for guinea pigs. Each calf received a subcutaneous injection consisting of 10 cc of a saline suspension from a 48-hour agar culture. The suspension was approximately five times as dense as tube 1 of the McFarland scale. In a similar manner, the second injection was administered six weeks later.

The heifers were bred when 12 to 14 months of age to a bull negative to successive agglutination tests. Regular monthly agglutination tests were conducted according to the technic recommended by the United States Live Stock Sanitary Association. Exposures were given by the mouth to a recently isolated bovine *Brucella* strain when the heifers were between five and six months in gestation.

### TREND OF THE SERUM AGGLUTINATION TITRES

Six weeks following the preliminary injection, maximum agglutination titres of 1:500 were obtained in three heifers. Maximum titres of 1:1,000 were observed in two heifers subsequent to the second injection. Ten months following the second calfhood injection, before exposures were given, four heifers were negative to the agglutination test. Two of the four remaining heifers showed complete agglutination in titres of 1:500, one of 1:100 and one of 1:50. Following the exposures, two heifers developed titres of 1:1,000, two of 1:100, and two of 1:25. The sera of the two remaining heifers remained negative. At parturition

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five heifers showed a rise in titre, the highest reaction occurring in a 1:2,000 dilution. Subsequently, the titres dropped except in one heifer that showed a titre of 1:2,500 two months following parturition. The titre of this animal gradually declined to 1:500. Two controls developed titres of 1:2,500 following exposure. The third control gave a maximum titre of 1:500.

#### RESULTS OF FIRST PREGNANCIES

The eight injected heifers dropped living calves at full term. One calf died at birth and one lived for about twelve hours. *Br. abortus* was not recovered from the tissues of either calf but was found in the placentae of the dams. Three of the six remaining heifers were found harboring *Br. abortus* either in the uterus or udder. The agglutination test of the milk serum of two heifers was negative. One heifer showed incomplete agglutination of the milk serum of one quarter in the 1:50 dilution, and in another heifer the reaction was incomplete in a dilution of 1:100. Complete agglutination of the milk serum from each quarter in a 1:100 dilution was found in one heifer, and another heifer had a 1:100 titre in two quarters.

Two controls aborted and one dropped a living calf. *Br. abortus* was recovered from the uterus and udder of the two heifers that aborted and from the uterus of the heifer that gave birth to a live calf. The milk serum of two controls gave complete agglutination reactions in a dilution of 1:100 and in the third control the reaction was complete in the 1:1,000 dilution.

#### APPLICATION OF THE OPSONO-CYTOPHAGIC TEST

The phagocytic power of the blood following parturition was determined according to the method described by Huddleson.<sup>1</sup> The degree of phagocytic activity of each heifer is indicated in table I.

Four heifers showed some polymorphonuclear cells containing more than 50 bacteria per cell. Only one heifer had more than 50 per cent of the cells showing marked phagocytic activity. Some of the vaccinated heifers did not show any more activity than the controls.

The calves of six vaccinated heifers were found to have only a slight phagocytic activity two months following delivery. In one calf 20 per cent of the cells showed marked activity. There was marked activity in 12 per cent of cells of another calf. The blood of the live calf of one control did not show any marked phagocytosis.

TABLE I—Calving record, serological and cultural findings, and record of opsono-cytophagic test.

HEIFER	PARTURITION		MAXIMUM AGGLUTINA- TION TITRE FOLLOWING EXPOSURE	BRUCELLA ABORTUS INFECTION		OPSONO-CYTOPHAGIC TEST									
	DATE (1935)	CALF		UTERUS	UDDER	DATE (1935)	M <sub>A</sub>	M <sub>O</sub>	S	N	DATE (1935)	M <sub>A</sub>	M <sub>O</sub>	S	N
Vaccinated	133	3-13	Live	—	+	6-20	0	0	3	22	12-2	2	1	2	20
	135	3-24	Live*	+	+	6-20	0	2	6	17	12-2	0	0	0	25
	140	3-8	Live*	+	+	5-21	4	7	13	1					
	141	4-15	Live	—	+	6-20	0	0	1	24	12-2	0	0	0	25
	142	4-9	Live	+	—	6-20	1	1	4	19	11-5	0	0	0	25
	144	3-25	Live	—	—	5-21	14	9	1	1					
	148	3-10	Live	+	—	6-20	0	0	5	20	11-5	0	1	2	22
	150	4-4	Live	—	—	6-20	0	1	5	19	11-5	0	0	0	25
Controls	139	4-9	Aborted	+	+	5-21	0	1	12	12	12-2	0	0	2	23
	143	3-4	Live	+	—	6-20	0	0	1	24					
	147	2-8	Aborted	+	+	5-21	0	4	7	14					

\*Died shortly following delivery.

Key: Degree of phagocytosis:

Ma = marked—over 40 bacteria per cell.

Mo = moderate—21 to 40 bacteria per cell.

S = slight—1 to 20 bacteria per cell.

N = negative—no bacteria in cells.

+ = *Br. abortus* isolated.— = *Br. abortus* not isolated.

## DISCUSSION

Although most of the heifers in this experiment were given injections when two to three months older than those in a former experiment,<sup>2</sup> the trend of the agglutination titres was comparatively the same. The titres dropped appreciably four months following injection. As was the case in the former experiment, one heifer (141) showed only a slight agglutinin response to the calfhood injections. The blood serum became negative to the agglutination test and remained negative even following exposure during pregnancy. It was interesting to note that the phagocytic power of the blood of this animal was low.

It appeared that a placental resistance to *Br. abortus* infection was developed as indicated by the number of living calves obtained. The bacteriological examination of the tissues of two calves, shortly after they were born, did not reveal *Br. abortus* infection. *Brucella* organisms, however, were obtained from the placentae of the dams.

A considerable degree of phagocytic activity was obtained with the blood of only one heifer (144). Since the phagocytic power of the blood was not determined either following the calfhood injections or during pregnancy, it is possible that more activity existed earlier and disappeared in a short time.

Huddleson<sup>3</sup> has recently reported that the opsono-cytophagic activity in vaccinated herds indicated that active immunity was of short duration in a number of animals in each herd. An improved method for estimating the opsono-cytophagic activity has been developed recently by Huddleson.<sup>4</sup> It seems that the study of the phagocytic power of the blood in an opsono-cytophagic system offers a possibility of evaluating more accurately the relative merits of various procedures to develop resistance against *Brucella* infection.

## SUMMARY

Eight heifer calves that received two subcutaneous calfhood injections of a living *Brucella abortus* culture dropped living calves. Two calves died shortly following delivery. *Br. abortus* infection was recovered from five heifers.

Serum agglutination titres, subsequent to the administration of two injections, were higher than when only one injection was given. The titres dropped considerably four months following the second injection.

A study of the opsono-cytophagic activity of the blood following parturition revealed only a slight degree of phagocytosis in most cases.

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## OAT HAY POISONING\*

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A loss of 67 head of registered cattle near Franktown, Colorado, on March 31, 1936, following the consumption of oat hay, has given this opportunity to carry out some investigational work and to survey some previously reported outbreaks. Whether oat hay is ever in itself poisonous or is injurious only through some other constituent has not been determined, but some light has been thrown on the problem and since there is so little in the literature on the question, it seemed best to record the findings.

### PREVIOUS OUTBREAKS

On March 16, 1923, near Kiowa, Colorado, 125 head of Short-horn cattle were fed oat hay after a light snow had fallen. Within the next four hours, 41 head died. The owner reported that the animals craved water and most of them died soon after drinking. Three-fourths of this stack had been fed to the same cattle prior to the outbreak. The oats had been cut just as they were heading out and showed some rust. The predominating weeds were pigweed and lakeweed. The weeds had not gone to seed. There was no unusual amount of mold present.

This outbreak was originally called to our attention by Dr. Chester Nelson, at that time practicing at Kiowa. Some days later, the place was visited by Dr. G. W. Stiles and the senior author, but the carcasses had all been disposed of and nothing significant could be discovered in the hay.

On December 24, 1923, 21 head of cattle died after eating oat hay on the ranch near Franktown mentioned in the introduction and where the recent loss of 67 head took place. This matter was called to our attention by Dr. C. W. Converse, the practitioner there, and on January 1, Dr. W. E. Howe, of the U. S. Bureau of Animal Industry, and two of the present authors visited the place. The carcasses were still available but were thoroughly frozen so that a satisfactory examination was impossible. The oat hay appeared to be of unusually good quality, was bright, not moldy and contained only a comparatively small number of weeds. Pigweed was the most prominent. Later in the spring,

\*Received for publication, July 29, 1936.

the owner ran short of feed and fed out this stack with no bad results.

On April 10, 1935, a man near Craig, Colorado, lost three two-year-old cattle soon after the consumption of oat hay that was damp from melting snow. He later fed this hay to horses with no bad results.

Dr. Converse reports two additional outbreaks that came under his observation, one in which either four or five calves died and another in which 18 head died after eating oat hay.

Dr. O. A. Beath, of the University of Wyoming, in a personal communication, tells of two such outbreaks in northeastern Wyoming in one of which 44 head died. Of the remaining animals several aborted within the next few days, a circumstance that was not mentioned in previous outbreaks. In one of these outbreaks oat hay seemed to be the offending substance but in the other it was oat straw.

Dr. H. D. Port, state veterinarian of Wyoming, tells of having his attention called to at least seven outbreaks of similar character during the past winter. Probably two of them were those mentioned by Dr. Beath. In all of these cases either oat hay or oat straw was under suspicion.

Rogers and Boyd,<sup>1</sup> in a discussion of "Sudan Grass and Other Cyanophoric Plants," quote a letter from an owner as follows:

Recently I began feeding hay composed of Canada thistle and oats and since that time I have had two sick cattle. Altho I am not certain, I believe the hay was responsible for the trouble. I am wondering if some of your staff could advise me how (if possible) I can utilize the rest of this hay, for I need it for feed and I have three or four tons of it.

This hay does not seem to be spoiled altho it does have a sort of "musty" smell. It was impossible to cure this hay properly last summer, because of the large proportion of thistles (about 50%) and because the oats were not yet headed out. It is slightly damp, though not wet, and I find traces of mold on some of the thistle stems and on some of the joints of the oat stems.

Regarding the animals, I would say that they became sick quite suddenly, a few hours after eating the hay, and that they recovered rapidly. While in their worst stages the animals were prostrate on the floor and their breathing sounded like hiccoughs or grunts.

They state also that other deaths from eating Canadian thistles and oats are included in their series but that in no such case could cyanide be demonstrated.

#### THE PRESENT OUTBREAK

The immediate outbreak was called to our attention by Drs. Francis and Stiles, of the U. S. Bureau of Animal Industry, and Dr. Converse, local practitioner, to whom we are indebted for

close coöperation. They examined the carcasses of several of the animals, assisted in the investigation and furnished much of the information here presented.

Two lots of Hereford cattle were kept on this place, one comprising 30 fresh cows and another consisting of 104 mature animals and 46 calves. All of these had been carried through the winter on cane hay, corn fodder and some cotton cake until four days previous to the outbreak, when they had been placed on the oat hay but continued on the cake. Approximately half of the stack had been fed out.

About 1:30 p. m. on March 31, a load of the oat hay was hauled to the first lot of cows. At the time a light snow was falling. At 4 p. m., one of these cows sickened and Dr. Converse was called. He treated the cow and she recovered.

About 2:30 the same day, a load of the same hay was fed to the other lot. The snow was increasing in intensity. Within two hours several animals sickened and, by 7 p. m., 67 head had died. All were mature cows except one bull and one eight-month-old calf. All animals that showed symptoms died with the exception of six or eight. Six of these dropped fully mature dead calves within the next few days. The herd had been tested for Bang's disease and no abortions had occurred previously.

#### SYMPTOMS

The animals showed trembling, weakness, staggering gait, finally falling without convulsions. A few showed a desire to fight and one charged an attendant and fell dead. Some gasped for breath and frothed at the mouth. After the animals went down, however, the breathing was not noticeably increased but the mouth might be open. The pulse was rapid and weak. Cyanosis was marked on the sclera, the muzzle, the tongue and the udder. The temperature was normal or subnormal.

As soon as it was apparent that the oat hay was dangerous, the remainder was gathered up from the ground where it had been fed and thrown onto the butt of the cane stack in the stack yard. Some of it remained on the ground, however, and was later consumed by horses without ill effect.

#### THE OAT HAY

The hay had been raised on the ranch in the creek bottom, had been cut about August 3, when still immature, although headed out and stacked August 14. The chief contaminant was pigweed (*Amaranthus retroflexus*), although there was some Russian thistle, sunflower, sedge and wild morning glory. The

hay was of good quality but felt damp when the hand was thrust into the stack. It was only slightly moldy. A few of the heads were smutty.

Samples of the hay that had been returned to the stack yard after the death of some of the cattle were submitted to Prof. J. W. Tobiska, chemist for the experiment station. By the application of the sodium picrate paper test,<sup>2</sup> he reported hydrocyanic acid in a composite sample. It was found on examination, however, that some cane hay was present in the sample. When the various constituents were sorted and tested individually, the oats, sedge, sunflower, pigweed and morning glory gave negative tests and only the cane was positive.

#### TESTS WITH LABORATORY ANIMALS

Two samples of hay, one taken from the top of the stack butt and another from deeper in, were submitted. They differed chiefly in the moisture content, the deeper one being damp. Both samples were subjected to the same treatment. First the hay was ground finely and 30 gm of each sample was placed before two guinea pigs. They ate very sparingly until it was mixed with ground barley, when it was all consumed. The pigs remained normal. Similar amounts of each sample were fed to a rabbit and with the same result. The experiments were then repeated with both guinea pigs and rabbits except that 60 cc of distilled water was added to each sample of ground hay. The results were negative.

Samples to which the distilled water was added were then allowed to stand in sealed jars at room temperature for 21 hours, then in the 37° C. incubator for an additional five hours, and then fed to rabbits and guinea pigs with no bad results.

Fifty gm of each hay sample was soaked in 350 cc of sterile .85 per cent salt solution in a closed jar in the refrigerator for 2½ hours, then successively passed through absorbent cotton, a Seitz clarifying disc and a Mandler candle, thus rendering the filtrate sterile. Five cc of the filtrate was then given intraperitoneally to a guinea pig with no bad result. The experiment was repeated only with the material from the center of the stack, using 25 gm of the hay and 250 cc of the salt solution and allowing it to stand for 2½ hours at 37° C., giving 5 cc of the filtrate but with the same result.

Then twelve gm of the smutty oat-heads were picked out and soaked in 120 cc of salt solution for 2½ hours in the refrigerator and then filtered as above. Five cc of the filtrate was injected intraperitoneally into a guinea pig but with no deleterious result.

## PIGWEED EXPERIMENT

A quantity of the pigweed was sorted from the oat hay and ground. A portion submitted to Prof. Tobiska of the chemistry section was reported as negative for hydrocyanic acid. Fifty gm was soaked in 300 cc of .85 per cent salt solution in a sealed fruit jar in the refrigerator overnight. A rabbit and a guinea pig were each given 9 cc by the mouth with no significant result.

Extract prepared in the same way then was passed through the Seitz clarifying disc and the Mandler filter and 10 cc and 5 cc, respectively, administered intraperitoneally to a rabbit and a guinea pig. No untoward results followed.

Then 25 gm was extracted with 104 cc of ether under the conditions already described. The ether was allowed to evaporate and the material resuspended in 10 cc of physiological salt solution. A rabbit and a guinea pig each received 5 cc of this preparation by the mouth with negative results.

Next 25 gm was extracted with 100 cc of ethyl alcohol as described above, the alcohol allowed to evaporate at 37° C. and the residue redissolved in 10 cc of salt solution. A rabbit and a guinea pig were each given 5 cc of this by the mouth without significant result.

## FEEDING EXPERIMENTS WITH CATTLE

Six yearling steers were purchased in the Denver stock yards. They were of mixed dairy type, of probable southern origin, and weighed approximately 500 pounds each. With the exception of small abscesses on the jaws of two of them, they were apparently healthy. They were trucked to the Grant ranch on Saturday, April 18. The steers were placed in a dry corral and feed was withheld but water allowed until the experiment was begun two days later. The steers were divided into three lots.

*Lot 1:* Steers 1 and 2 were fed oat hay from the top of the stack, that contained a small quantity of cane hay that had become mixed with it in handling. The amount of cane would probably not exceed 1 per cent. This hay was moistened with water and fed at 10:15 a. m.

*Lot 2:* Steers 3 and 4 were fed oat hay that contained no cane. It was taken from the top of the stack and had been exposed to the air and sunshine. It was moistened and fed at 10:15 a. m.

*Lot 3:* Steers 5 and 6 received hay from deep in the stack where it had not been exposed to air or sunshine. It was cold and clammy but not wet and had a slight musty odor. There was no evidence of molds, but smut was observed in some of the

heads of the oats. This lot was fed at 10:30 a. m. It was intended that this hay be fed just as taken from the stack but rain began falling soon after noon and most of it was consumed while wet. None of the steers seemed accustomed to being fed hay and ate rather sparingly for a time, but in the course of the day a considerable amount was consumed. They were all allowed access to water at noon and drank freely.

No symptoms developed until 8:15 p. m., when steers 1 and 5 were taken suddenly ill and died within half an hour. Steers 2 and 6 also developed alarming symptoms but recovered during the night.

The next morning at 8:30, steers 2 and 3 were fed hay from deep in the stack the same as steers 5 and 6 had been fed the previous day. The day was bright and the hay remained dry throughout the day. There were no apparent bad effects upon these animals. Steers 4 and 6 were fed hay from the top of the stack. This hay was moistened and fed under a shed, where it remained moist throughout the day. Steer 4 sickened about noon and died at 1:10 p. m.

#### SYMPTOMS

The symptoms of steers 1 and 5 were not observed by us, as we had left the place before they became ill. As reported by the attendants, they showed short, irregular breathing, a humped-up appearance, trembling, weakness, staggering, blindness and falling without much struggling. One of the animals fell into the fence.

Steer 4 stopped eating, became dull, humped up, head down, staggered when forced to move, went down about a half-hour before death, but lay rather quietly on its side. The mouth was open at times and the eyes rolled. There was, however, no gasping for breath. The respiration was 16, the temperature 98.8 and the pulse 120, weak and irregular. Cyanosis was noted in the sclera and tongue. Death took place without struggling.

#### POSTMORTEM EXAMINATION

*Steer 1:* A little froth appeared at the nostrils. The nose and scrotum were bluish. The blood was dark red but turned brighter on exposure to the air. It was very poorly coagulated except in the largest vessels near the heart.

The right lung showed hypostatic congestion. The tracheal mucous membrane was studded with hemorrhages. Petechiae were present in the epicardium over the ventricles. The endocardium was pinkish in color. The spleen, liver, gall-bladder and kidneys

were apparently normal. The urinary bladder showed capillary congestion.

The anterior cul de sac of the rumen showed deep reddening after the epithelial layer was removed. The mucous membrane of the abomasum was bright pink in color. The contents of the posterior portion of the duodenum were stained with blood. The walls were interlaced with hemorrhagic streaks. In portions of the jejunum the same condition prevailed. The cecum showed numerous hemorrhages.

*Steer 5:* Similar conditions prevailed as in steer 1, except the walls of the rumen showed a little more and the lungs a little less congestion.

*Steer 4:* Posted within an hour after death. The most striking phenomenon was the chocolate brown color of the blood that gave all of the organs containing much blood the same brownish appearance. This was noticed especially in the spleen, liver, heart, kidneys and lungs. The blood had not coagulated but did so on exposure to air. The rumen did not show the reddening of the wall as in the other two. The mucous membrane in the anterior portion of the abomasum was rose colored. All other organs were normal.

#### EXAMINATION OF THE RUMEN CONTENT

Early the next morning, after the death of steers 1 and 5, sodium picrate papers were placed in tubes open at the lower end. Then a tube was inserted into a hole made through the abdominal wall so that the gases of the rumen came in contact with the papers. Within 30 minutes, both steers gave a distinct reaction for hydrocyanic acid which deepened considerably in the next two hours. The same test applied to steer 4 was negative at the end of 15 minutes.

Ruminal content from all three animals was placed in glass fruit jars, sealed and taken to Fort Collins. Prof. Tobiska reported negative for hydrocyanic acid, using the sodium picrate paper test.

To 50 gm of each sample was added 50 cc of .85 per cent salt solution, the mixture placed in a sealed jar and refrigerated for one hour, and then passed through the Seitz clarifying disc and the Mandler candle. Five cc of each filtrate was given intraperitoneally to a guinea pig with no harmful result.

#### CONTINUATION OF THE CATTLE EXPERIMENT

The three remaining steers then were placed on cane hay for twelve days. Two of them received, in addition, salt to which

had been added  $7\frac{1}{2}$  per cent sulfur. The other did not receive the sulfur salt. This was done to test the South African finding<sup>3</sup> that sulfur was protective against hydrocyanic acid. At the end of this time, the three steers were given liberal feedings of moistened oat hay from deep in the stack. All remained normal.

#### THE SHEEP EXPERIMENT

The steers then were sold and twelve ewes were purchased. They were fasted for 24 hours and then sorted into four lots of three each.

*Lot 1:* Fed oat hay from deep in the stack in open corral. Hay was moistened and became quite wet later in the day as a result of rain and snow.

*Lot 2:* Fed hay from same source as lot 1. Fed under shed and remained dry throughout the day. A bundle of hay from same source as above was sorted carefully, and the weeds separated from the oats.

*Lot 3:* Fed the oats moistened.

*Lot 4:* Fed the weeds moistened.

The weeds were almost entirely pigweed. There was a small amount of wild sunflower.

The day was wet and cold and the ewes stood with their backs arched and shivered. They did, however, take to the feed readily and were not long in taking on a good fill. Water was allowed but taken sparingly. No abnormal symptoms were observed at any time.

They were kept entirely on this oat hay for 28 days, when they were brought to Fort Collins by truck. However, some of the hay was brought with them and they continued to eat it for two or three days longer. No illness was noted at any time.

#### DISCUSSION

The evidence that plants such as oats, thistles, flax, millet and various weeds usually not considered to be cyanophoric, when made into hay, are at times dangerous is too strong to be longer ignored. In all instances previously reported the suspected forage was fed with impunity when subjected to experimental trial. In several such instances the incriminated hay was fed out later to cattle with no loss. In those cases where horses and sheep ate the same forage, no loss was sustained in these animals.

Since pigweed, in this western country at least, is usually the chief contaminant of oat hay, it is only natural that suspicion should attach to it. In 1927, a large number of cattle died in eastern Colorado as a result of eating the green plant. An un-

usual situation prevailed that year. Late summer rains brought up a luscious stand of that weed and since the grass was short the cattle ate it greedily. Investigation at that time led to the conclusion that the deaths were due to bloat and not to any poisonous substance in the plant itself. Nothing reported here indicates that it was the offending agent in the oat hay outbreak described.

Moisture seems to play some part, since in several of the outbreaks it was reported that a light snow fell on the forage. In our experiments with cattle it was felt that moistening the hay was a significant factor in reproducing the symptoms.

Whether the positive test for hydrocyanic acid obtained in steers 1 and 5 is significant cannot be determined at this time. It is even possible that a positive test would have been secured in the third steer if more time had been given. The situation is still further complicated by the fact that the only constituent of the hay samples that gave a positive test was the cane and yet the animals had been eating cane hay for months with no bad results and were changed back to it with safety. There seems to be almost no possibility that cane could have caused this loss.

It must be admitted that the symptoms are those of cyanide poisoning, but the proof is far from satisfactory. The Minnesota workers are even skeptical of the lethal factor being hydrocyanic acid in well-known cyanophoric plants such as Sudan grass. If not, then a poison acting similarly and present in a variety of forage plants remains to be discovered. The manner of its release should also be investigated although moisture as stated above may play a part.

The occurrence of abortion in six of the recovered animals, coupled with a similar report in one of the Wyoming outbreaks, deserves further attention.

#### SUMMARY

Several serious losses of cattle feeding on oat hay are described. The symptoms are suggestive of poisoning by hydrocyanic acid, yet no cyanide has been found in the hay except in cane, which in most of the outbreaks is not even under suspicion.

Pigweed (*Amaranthus reflexus*) is almost always a contaminant of oat hay but produced no disturbance in sheep, rabbits or guinea pigs when fed experimentally.

By feeding the original hay the disease was apparently reproduced in five of the steers, causing death in three. Two of the three showed the presence of hydrocyanic acid in the rumen by means of the sodium picrate paper test, some hours after death.

No injurious substance could be shown to be present in the hay or any of its constituents when fed to sheep, rabbits or guinea pigs.

Watery, alcoholic and ether extracts were harmless to laboratory animals.

## REFERENCES

<sup>1</sup>Rogers, C. F., and Boyd, W. L.: Sudan grass and other cyanophoric plants as animal intoxicants. *Jour. A. V. M. A.*, lxxxviii (1936), n.s. 41 (4), pp. 489-499.

<sup>2</sup>Henrici, M.: Preliminary report upon the occurrence of hydrocyanic acid in grasses of Bechuanaland. 11th & 12th Rpts. Dir. Vet. Ed. & Res. (1926), p. 495.

<sup>3</sup>Steyn, D. G.: Prevention of prussic acid poisoning. *Onderstepoort Jour.*, iii (1934), pp. 433-434.

## PERSONALS

DR. OTTO HORNLEIN (McK. '10) retired from active service in the U. S. Bureau of Animal Industry on October 31, 1936. He had been in the service over 30 years. At the time of his retirement he was in charge of meat inspection at South Saint Paul, Minn.

DR. J. P. BUSHONG (U. P. '06), of Los Angeles, Calif., for many years connected with the Los Angeles County Medical Milk Commission, has resigned to take the position of City Veterinarian and Director of Milk and Meat Inspection for the City of Los Angeles.

DR. EDWARD L. VAIL (U. P. '36), who has been connected with the laboratory of the Pennsylvania Bureau of Animal Industry the past year, has accepted a position with the U. S. Bureau of Biological Survey, and is now stationed at Los Angeles as agent-in-charge.

DR. O. H. MUTH (Mich. '29), who has been at the Oregon State College and Experiment Station, Corvallis, for a number of years, has resigned his position there to accept appointment as veterinary pathologist in the Department of Agriculture and Markets, Madison, Wis.

DR. J. G. HARDENBERGH (U. P. '16), of Plainsboro, N. J., was elected president of the International Association of Milk Sanitarians (formerly the International Association of Dairy and Milk Inspectors) at the 25th annual meeting, held in Atlantic City, N. J., October 14-16, 1936.

DR. J. F. SHIGLEY (Corn. '15), of Pennsylvania State College, was recently elected a member of the Board of Trustees of the Center County Hospital. Dr. Shigley has served on the State College Board of Health for the past ten years and recently was reappointed to the Board.

DR. J. W. SCHNEIDER (Gr. Rap. '13), of Riga, Mich., has retired from practice and has gone to Georgia, where he owns a large acreage devoted to turpentine production and the growing of vegetables. His practice will be taken over by Dr. E. C. Heinsen (O. S. U. '31), of Port Clinton, Ohio.

DR. GEORGE A. FERGUSON (U. S. C. V. S. '20), of Leaksville, N. C., recently addressed the local Rotary Club on the value of a military company to a community and the splendid training which its members receive. Dr. Ferguson has commanded the local Headquarters Company, 60th Infantry Brigade, N. C. National Guard, for the past six years. Last summer the unit received high praise from the Regular Army officials.

167955

## A DISEASE OF HORSES CAUSED BY FEEDING MOLDY CORN\*

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Serious losses in horses have been recorded periodically for a number of years attributed to poisoning by moldy corn. This condition has been known as moldy corn poisoning, cornstalk disease, leukoencephalitis or acute hemorrhagic encephalitis and has often been confused with the specific equine encephalomyelitis caused by a filtrable virus.

The specific encephalomyelitis of horses caused by the various types of filtrable virus has been adequately described by a number of American investigators including Meyer, Shahan and Giltner, and Records and Vawter, who have established beyond any doubt the presence of the filtrable virus in such cases. In the summer and fall of 1912, Udall<sup>1</sup> made a careful study of an epizootic among horses in Kansas. Although he did not demonstrate the presence of a virus, his pathologic descriptions indicate that he was dealing with the virus form of encephalomyelitis. He reports no gross cerebral necrosis. Buckley and MacCallum,<sup>2</sup> in 1901, described a disease of horses in Maryland similar to that discussed in this report. They associated its occurrence with feeding moldy fodder. In 1902, Butler<sup>3</sup> produced a typical case of the non-virus disease showing cerebral liquefaction by feeding moldy ear corn to which were added chaff and residue blown from the moldy corn by the sheller. About four weeks were required to produce the experimental case. The recent report by Doyle<sup>4</sup> indicates some confusion regarding lesions similar to those of moldy corn poisoning and those of the specific virus encephalomyelitis. Perivascular cuffing and neuronophagy were not present in the cases of moldy corn poisoning which were studied by us.

In Iowa, during the winter of 1934 and spring of 1935, many horses which succumbed manifested symptoms indicating a severe central nervous disorder and consequently the cases were diagnosed clinically as specific virus encephalomyelitis. Examination of the brains of these horses failed to show any evidence of the virus disease, either by microscopic examination or by the experi-

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mental inoculation of animals. The brains of horses received during the winter and spring invariably showed disintegration and liquefaction in the white substance of the cerebral hemispheres. In several instances, only the gray substance remained intact, forming a shell about the disintegrated white brain tissue. One horse which showed severe clinical symptoms was destroyed. The brain showed a spherical lesion about one inch in diameter in the base of the brain. This area appeared edematous and was of a yellowish-orange color.

During the months of July, August, September and October, however, the brains of the horses sent to the laboratory from the field failed to show this characteristic liquefaction. Microscopic examination of this material did show typical perivascular cuffings and neuronophagy which are invariably present in the virus disease. The findings in this group of cases are compatible with the epizootology of the virus disease, which subsides after one or more killing frosts. The chart in figure 1 shows the seasonal occurrence of the two conditions.

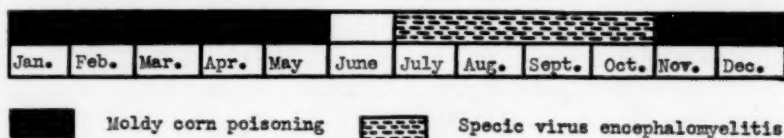


FIG. 1. Chart showing seasonal occurrence of moldy corn poisoning and encephalomyelitis.

The first cases encountered showing liquefaction necrosis in the brain tissue were received in November. This condition most commonly occurred in areas where moldy corn was being harvested. This history of these cases indicated that in some instances the horses had been fed hay and oats but had eaten considerable of the moldy corn while being used on the wagons in the field. In other instances horses which were turned into the corn fields died. Later we reproduced this condition in horses experimentally by feeding moldy corn and corn fodder.

Some of the material which was sent to us from the field for examination proved to be contaminated. Cultures of *Escherichia coli* were often obtained from the brain tissue. Diplococci were cultured from several cases sent in from the field. Some of the fresh material proved to be free from bacterial infection and the material taken from our experimentally produced cases failed to show the presence of any bacterial contamination. No virus could be demonstrated in the brains of experimentally fed horses that

died as the result of consuming moldy corn. When material taken from the liquefied areas of the brain was injected intracerebrally into guinea pigs, death sometimes resulted within 6 to 8 hours. When material was taken from the non-liquefied regions and likewise injected, no reaction was obtained. This evidence indicated that we were not dealing with the specific virus disease.

#### EXPERIMENTAL

The animals used in this experiment were in good condition and apparently normal. Five horses were placed in a dry lot and fed moldy corn and a good grade of corn fodder. Three horses, used as controls for this experiment, were fed clean corn and corn fodder. The corn selected for this purpose was brought to the Research Institute from a farm located in a region where heavy losses occurred during the month of November. This lot of corn was purchased on March 30 and was in much better condition than much of the corn that was fed to horses during the previous month of November. All of the horses which were fed this moldy corn developed well-defined clinical symptoms. Those which died showed extensive brain lesions, chiefly areas of liquefaction necrosis, edema and hemorrhage in the white brain matter. Many of these necrotic areas were of a creamy consistency and were yellowish-orange in color. Congestion and hemorrhage could often be observed in various regions of the white matter. None of these cases showed lesions such as those associated with the specific virus encephalomyelitis. The histopathology consisted of liquefaction necrosis, edema, extensive hemorrhage and degenerative changes in the brain.

The control horses which were fed clean corn and the same good grade of corn fodder in the experimental feeding of the five horses showed no clinical symptoms or any untoward response. They were released in good condition after three of the five test horses died and the two others showed severe clinical symptoms. The following protocols will show the development, course, clinical symptoms, and postmortem findings of the test horses.

#### HORSE 755

- April 2—Fed moldy corn and good grade corn fodder; condition and appetite good.
- May 11—Appetite diminished and slightly depressed.
- May 12-21—Appetite good, apparently normal in all respects.
- May 22—Depressed; slight incoördination; tendency to drag right hind leg while running.
- May 25—Considerably depressed; crossing front legs at times while standing; severe incoördination.

May 26—Subject walked in circles, crashing into the fence as if blind. The horse was soon down on left side, thrashing around. Temperature 101. Died about noon.

*Postmortem findings:* The liver, kidney and bladder showed evidence of a severe toxemia. The bladder, ureter and renal pelvis contained a yellowish-orange colored sero-mucous exudate. The liver appeared swollen and congested. The lobules presented a yellowish-orange color.

Left cerebral hemisphere partially collapsed upon removal from the cranial cavity. It fluctuated upon pressure. The right cerebral hemisphere appeared firm. Upon incision, the left hemisphere revealed advanced liquefaction necrosis of the white matter. In this soft, creamy, disintegrated brain tissue numerous fragments of the greatly degenerated vascular system could be seen. Figure 2 shows the extent and nature of this necrotic area.



Fig. 2. Brain showing extensive necrosis and hemorrhage in left cerebral hemisphere.

*Microscopic examination:* Extensive necrosis present, in which many blood-vessels and capillaries could be seen. Many of the vessels were surrounded by blood. In the tissue adjacent to the liquefied area extensive edema and hemorrhage could be seen. Small vessels containing many leukocytes were rarely encountered. The characteristic perivascular cuffing typical of virus cases was not observed. The glial elements showed an increase in the tissue which was not necrotic. The right cerebral hemisphere which showed no gross necrosis showed microscopic hemorrhages, slight congestion and a mild glial response.

The liver showed a slight cellular hepatitis with hemochromatosis and advanced cloudy swelling. Epithelial degeneration was confined largely to the central region of the lobules. The spleen appeared severely congested with considerable hemochromatosis. The glomeruli of the kidney were congested and epithelial degeneration was evident in the cortex and medulla. The epithelium of the larger collecting tubules, calyces and pelvis, was vacuolated. In the lumen of the tubules

a mucoid substance, some of which contained pigment and fragments of blood-cells, could be observed.

## HORSE 753

March 31—Fed moldy corn and corn fodder; condition and appetite good.

April 17—Slight incoördination observed.

April 30—Quite depressed; appetite diminished.

May 1—Subject down on side most of the day.

May 2—Again up on feet and much brighter; incoördination progressively increasing.

May 6—Down; unable to rise; violent periodic movement of legs and head.

May 8—Horse found dead.

*Postmortem findings:* The left cerebral hemisphere showed extensive liquefaction necrosis which was limited to the white matter. There were no gross necrotic lesions in the right cerebral hemisphere. Gross changes found in organs similar to those of case 755.

*Microscopic examination:* Changes identical with those described in case 755.

## HORSE 754

March 31—Fed moldy corn and corn fodder.

April 28—Slightly depressed; appetite decreased.

April 29—Down.

April 30—Again up on feet; appetite fair; slight incoördination.

May 1-8—Progressive incoördination.

May 12—Down; arose with difficulty; incoördination considerably increased; tendency to walk in circles to the left. When down was stretched out with head flat on ground. Conjunctiva yellowish-gray; buccal mucosa gray and slightly icteric.

May 13—Horse on feet again; appetite fair; only slightly depressed.

May 14—Brighter; appetite good.

May 15—Quite active; only slight incoördination.

May 16—Again depressed and marked incoördination.

May 22—Down; unable to rise. Intermittent respirations. Temperature 98.5.

May 23—Moribund condition; animal destroyed.

*Postmortem findings:* No gross liquefaction necrosis. Near the brain stem several areas were observed which were slightly gelatinous. Gross appearance of liver, kidneys and bladder similar to the previous cases.

*Microscopic examination:* Areas of congestion, edema and hemorrhage were found in the pons, also in the cerebrum and in the region of the hippocampus. No evidence of perivascular cuffing or neuronophagy were observed.

## HORSE 756

April 2—Fed moldy corn and corn fodder.

May 21—Somewhat depressed.

May 23-26—Decrease in appetite; more depressed; difficulty in locomotion.

May 27-30—No apparent change.

June 1—Severe depression.

June 2—Down; unable to regain feet.

June 3—Subject thrashed about considerably; able to hold ears erect and partially raise head. Frequent spasmodic jerking of head accompanied by running movements of the legs. Mucous membranes not icteric; respirations fairly regular. Could lie only on left side; when turned would immediately turn over on left side again.

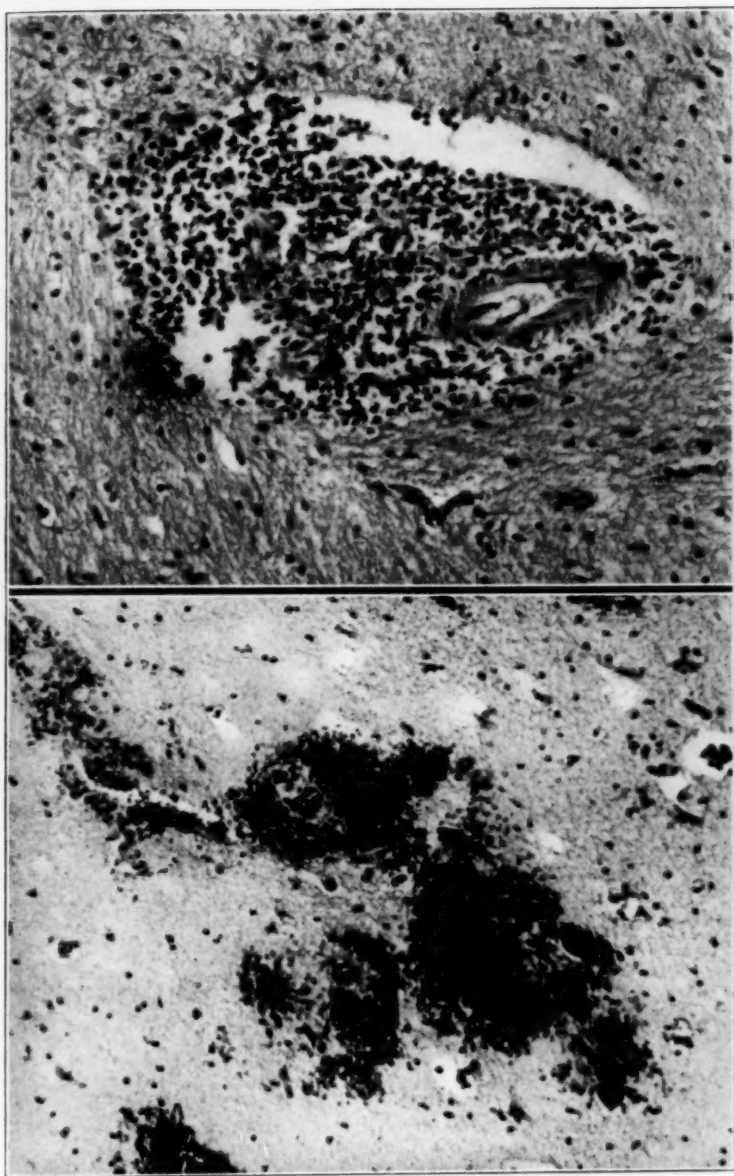


FIG. 3 (above). Brain from a specific case of virus encephalomyelitis, showing perivascular cuffing (x 200).  
FIG. 4 (below). Brain showing hemorrhage and necrosis from moldy corn poisoning (x 200).

June 4—Unable to rise to feet. Could raise head; drank three pails of water; movement of legs slower; respirations rapid and not so deep. Temperature 104.8; pulse 140; died in the night.

*Postmortem findings:* Liver, kidneys and bladder similar to previous cases; no definite gross liquefaction in the cerebral hemispheres; brain stem appeared congested and was somewhat soft. Spinal cord was considerably softened.

*Microscopic examination:* Liver, kidneys and spleen similar to previously described cases. Sections taken from the brain stem revealed considerable hemorrhage and degeneration. The degeneration had not advanced to a stage of liquefaction necrosis. Spinal cord showed advanced degenerative changes in the tracts and motor cells. Extensive hemorrhages were found also in horns. The severe lesions present in the spinal cord were sufficient to account for the incoördination.

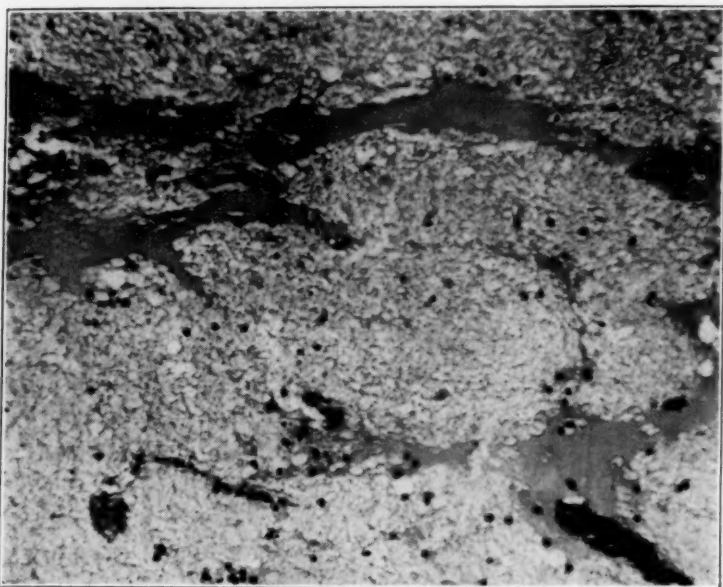


FIG. 5. Brain showing edema and necrosis from moldy corn poisoning (x 200).

#### HORSE 759

March 31—Fed moldy corn and corn fodder.

May 1—Slight incoördination; depression and decreased appetite.

May 2-10—Appetite and general condition seemed to improve periodically; slight incoördination.

May 11—Incoördination more evident; eye movement rather slow with eyelids relaxed showing considerable white of eyes.

May 25—Increased depression; progressive incoördination; swayed from side to side while walking; tendency to drag hoofs; hind legs were crossed at times.

May 26-July 15—Gradual loss of appetite with increasing emaciation. Progressive incoördination and depression. Temperature 101. Destroyed July 15.

*Postmortem findings:* Liver and kidneys similar to previous cases but not so much mucoid material in renal pelvis. No gross foci of liquefaction necrosis in the cerebrum. Medulla showed much congestion and some hemorrhage. Similar lesions were found in the base of the brain. There was considerable congestion and hemorrhage in the thoracic and lumbar regions of the spinal cord.

*Microscopic examination:* The pons, brain stem, cerebellum and spinal cord showed extensive hemorrhages and degenerative changes. The anterior horns of the spinal cord contained large hemorrhagic foci, which were apparently of longer standing. These contained pigment and the cells were undergoing hyaline degeneration.

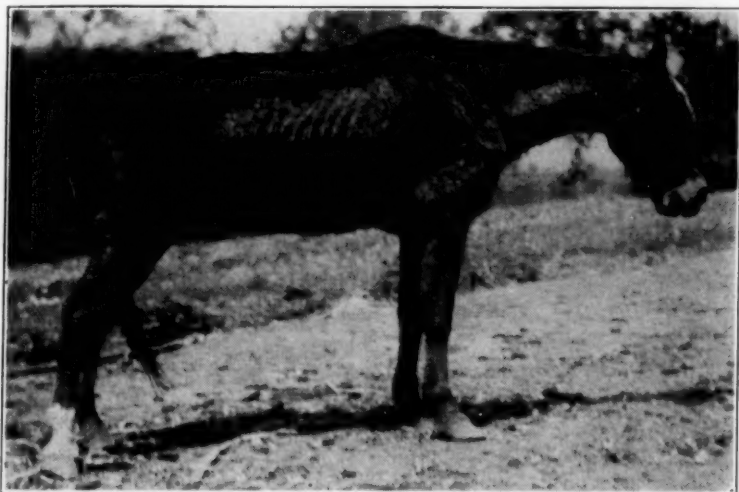


Fig. 6. Experimental moldy corn poisoning in horse, showing depression and marked incoordination. Note position of hind legs.

#### SUMMARY AND DISCUSSION

A disease of horses involving especially the central nervous system caused by the ingestion of moldy corn is described. This condition was produced under experimental conditions. Three of the five horses experimentally fed moldy corn died, while two were destroyed, one of which was in a moribund condition and the other showed marked intoxication and severe incoordination. These subjects presented gross and histologic evidence in the brain and spinal cord distinguishable from the specific virus disease. Bacterial cultures made from the brain and other organs were negative. Intracerebral inoculations into guinea pigs were likewise unsuccessful. This condition is not associated with the specific virus encephalomyelitis.

In Iowa, moldy corn poisoning occurs usually from November until May, during which time the virus disease has subsided. It

is possible, however, to have an overlapping in the occurrence of these two diseases in October and November, depending upon the temperature and rainfall affecting the development of the corn. The specific virus encephalomyelitis of horses is seldom encountered after the first killing frosts.

The perivascular cuffing and neuronophagy were never present in either field cases or those produced experimentally by feeding moldy corn. The moldy corn disease is always associated with necrosis, edema, advanced hemorrhagic lesions and degenerative processes. The two diseases were clearly differentiated on histologic evidence alone.



FIG. 7. Horse in moribund condition, from experimental moldy corn poisoning.

The term "moldy corn poisoning" should be used provisionally to designate this disease until the organism or group of organisms which are responsible for this action on the corn are isolated and their ability to produce these toxins demonstrated. There are a number of organisms present in moldy corn. Some are recognized as plant pathogens and others are not. Close cooperation with plant mycologists is necessary to determine the factors involved. None of these organisms which are found on moldy corn invades the animal body beyond the digestive tract. The term "cornstalk poisoning" is not well chosen as there is considerable evidence to show that the toxic agent is present in the kernels and possibly in the cobs as well. Because of the varied clinical pictures seen as the result of moldy corn poisoning, we do not feel justified in differentiating it clinically from the virus disease. The clinical symptoms and course of the disease depend largely on the extent and the part of the central nervous system which is involved. The history of the case and the clinical manifestations, together with the gross and histologic changes, should

make it possible to differentiate moldy corn poisoning from the specific virus encephalomyelitis in horses.

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- <sup>2</sup>Buckley, S. S., and MacCallum, W. G.: Acute hemorrhagic encephalitis prevalent among horses in Maryland. *Amer. Vet. Rev.*, xxv (1901), pp. 99-102.
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### Ohio Association of Public Health Veterinarians

Veterinarians engaged in public health work in Ohio have formed an organization known as the Ohio Association of Public Health Veterinarians. Dr. Warren P. S. Hall (Mich. '20), Chief, Bureau of Food and Drugs, Division of Health, Toledo, is president; Dr. L. C. Neer (O. S. U. '19), Division of Food Inspection, Board of Health, Middletown, is secretary-treasurer, and the following are members of the Executive Committee: Dr. H. H. Sparhawk (O. S. U. '08), Health Department, Akron; Dr. E. C. O'Dell (O. S. U. '07), Health Department, Columbus, and Dr. J. W. Burke (O. S. U. '23), Department of Public Welfare, Dayton.

### Visitors at the Journal Office

An unusually large number of out-of-town visitors called at the JOURNAL office during the past month. The number included:

- Dr. M. Barker, Regina, Sask.  
 Dr. A. E. Cameron, Ottawa, Ont.  
 Dr. Adolph Eichhorn, Pearl River, N. Y.  
 Dr. Emery H. Enge, Comfrey, Minn.  
 Col. Robert J. Foster, Washington, D. C.  
 Dr. J. C. Hargrave, Medicine Hat, Alta.  
 Dr. J. W. Harrison, Portland, Ore.  
 Dr. G. H. Hart, Davis, Calif.  
 Dr. A. C. Jerstad, Beltsville, Md.  
 Dr. Gordon C. Kendall, Lincoln, Ill.  
 Dr. B. J. Killham, East Lansing, Mich.  
 Dr. Fred W. Miller, Washington, D. C.  
 Dr. W. T. Oglesby, Baton Rouge, La.  
 Dr. J. D. Ray, Omaha, Nebr.  
 Dr. E. C. W. Schubel, Blissfield, Mich.  
 Dr. Cassius Way, New York, N. Y.

Everywhere in life, the true question is not what we gain, but what we do.—Carlyle.

## DISEASES OF THE DIGESTIVE TRACT OF CATTLE\*

*By J. L. AXBY, Indianapolis, Ind.*

I am fully cognizant of the scope of this title, and desire you to know the subject was assigned and not chosen. I am not unmindful of the fact that if so approached, provincial exigencies could be entertained and discussed leading to an unending discourse entirely separate and apart from fundamentals of anatomy, physiology and pathology. In the time allotted it becomes self-evident I must needs restrict this discourse to the diseases of the digestive tract of cattle commonly encountered in the field of every-day practice.

In the world's broad field of complex activities, great strides of progress have been made in preventive and therapeutic treatment in both man and the lower animals. In the medical profession the present heights of attainment have been reached from a common starting point of "First, I bleeds 'em, then I pukes 'em; then if they want to die, by God I lets 'em." In the veterinary profession our present attainments are in contrast with the days of "hollow horn," "wolf in the tail," and a greasy rag, or a piece of fat meat for "lost cud."

During the march of progress, we are profoundly grateful to the land grant colleges and universities, wherein departments and divisions were created, and administered by a competent personnel, qualified to experiment, carry out research, and make reports of findings, all of which make possible a more extensive and intensive live stock industry, creating a demand for qualified veterinary service, and brought into existence commercial laboratories from which other and new benefits are derived, to be passed on to the veterinarian for the mutual advantage of many persons and organized businesses, and thus add new modifiers to the social order of divisions of labor.

Personally I am proud to proclaim that we in Indiana recognize the refusal of Purdue's Chief Veterinarian, Dr. R. A. Craig, to have his activities and counsel subverted or directed in any way other than would square with the basic and fundamental intent in view, at the time of the Department's creation, and I hereby and now thank and congratulate him, and his corps of associates and assistants for the work they have done, for the ideals they possess, and for the coöperation they have given, and I am sure we are all happy to join with them in enjoying a greater per-

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\*Presented at the Purdue University Veterinary Short Course, LaFayette, Ind., October 21, 1936.

spective by reason of having attained a higher point from which to look in retrospect, and thus the better anticipate the future.

#### KNOWLEDGE OF PHYSIOLOGY ESSENTIAL

Experimentation in stomach digestion in bovines has led to definite findings in physiology, and consequently pathology is better recognized today than ever before. It must be recognized by all veterinarians that they cannot render competent and satisfactory service unless and until the complex physiology of bovine digestion is understood, which needs to be coupled to a reasonable understanding of feeds and feeding.

Without this preparation and a corrective, scientific following through, we can only anticipate, expect and deserve to have our passing characterized by being unwept, unhonored and unsung. I make this statement because I recognize it as being basic, and therefore in the beginning suggest a review at off times, an office postgraduate course, or attendance at every veterinary short course, seeking more light and the latest data on the subject from a standpoint of anatomy and functional use as compared to animals of other species.

#### IMPACTION OF THE RUMEN

One of the most common abnormalities is impaction of the rumen, which, as the term signifies, means that the rumen (or paunch) has become overloaded with food material. We think we are correct usually when we make such a diagnosis and proceed accordingly in our treatment.

We must not, however, lose sight of the fact that the stomach of the ox (in fact, of all ruminants) is affected by sensory reactions from within the body and also from without. There is much truth in the slogan, in part, "From contented cows," and it really means more than what may be facetiously said about "why" she is contented.

Any nervous irritation has its effect upon digestion, and any diseased condition characterized by pain tends to develop indigestion. Stomach indigestion in cattle primarily means a cessation of muscular movement, and care should always be exercised that we do not overlook the real source of the disturbance, as it may be found afar from the stomach itself.

The stomach of the ox has a dual sympathetic nerve supply, one increasing its motility and another that decreases its movements. This applies generally throughout the digestive tract, one nerve supply to accelerate movement, the other to inhibit movement.

I previously said that feeds and feeding need to be understood. For the sake of emphasis, I repeat it, for, in treating impaction of the rumen, we must give due consideration to the contents of the rumen from the standpoint of moisture content.

Stimulation of the accelerator nerves, to be expended on a dry stomach mass, is not only useless but harmful. Soften the mass, try to restore the stomach contents to a normal consistency first, then it will be reasonable to increase the enervation (accelerator nerve) and expect the muscular movement to handle food material.

#### THE IMPORTANCE OF WATER

The best vehicle of transportation throughout the alimentary canal is water, and any degree of inadequacy in moisture content at once invites derangement and disaster. Moisture originates from the water drunk, the saliva, and the feed itself. When indigestion sets in, the appetite is diminished, a cessation of saliva follows and the desire for water becomes diminished; therefore, often the patient is systematically dehydrated at the beginning of treatment.

The rumen should be well supplied with water, not risking voluntary drinking, but by using a stomach-tube, or forced drenching. However, I do want to impress on your minds, now and forever, that when pumping water through a stomach-tube into feed material in a dry, impacted rumen, it should be done slowly, and should always contain an antiferment. As soon as water contacts the warm food, where there is fermentation, unless the fermentation is expended throughout the food mass, it will be around the mass of food and tympanites may result and it may result very rapidly. Aromatic spirits of ammonia or thiosulfate of soda would fulfill the requirements as antiferments. Most veterinarians administer either magnesium sulfate or sodium sulfate; if such is done, it should be given greatly diluted. Palpation of the rumen will greatly assist in determining the fluidity of its contents.

When the contents have been suitably prepared for handling in a natural way, then we can select and administer that drug best suited to act as a stimulant to the vagus or accelerating nerve supply to the rumen.

Taking everything into consideration it appears to me that hypodermic administration would be more satisfactory than medication *per os*. Three-quarter-grain doses of eserine, arecoline  $\frac{1}{8}$  to  $\frac{1}{4}$  grain, and pilocarpine  $\frac{1}{2}$  to 1 grain it would appear, give the best response. Doses should be repeated to effect. Food

should be restricted in amount, sloppy or thin gruel, bran mash, and so forth, roughage withheld until rumination has returned.

#### CHRONIC OR PERSISTENT IMPACTION OF THE RUMEN

Occasionally we will discover what we might designate "a persistent impaction." This is one which refuses to respond to the usual treatment, with a history of having been "owner- or neighbor-treated," to no avail; no appetite, no thirst, no intestinal movement, with almost entire cessation of milk secretion. In these cases great care should be exercised that traumatic pericarditis be not overlooked, which, to me, can be decided with the use of a stethoscope in studying the heart action.

It having been determined that there is no pericarditis, then it resolves itself into liquifying the mass and breaking it down by controlled fermentation throughout the mass. Many times in these cases much water would be injurious; therefore, I would suggest more stress be placed on fermentation and recommend what would be equivalent to four cakes of yeast, dissolved in one pint of warm water every four hours, and kept up, even if pounds of yeast are given.

#### COLICS

Colic is an all-inclusive term, as it includes all pains existing in the abdominal cavity. What I mean is that attack which is seen in cattle characterized by every symptom of acute pain, such as that displayed by horses with acute colicky pains.

In treating these we should first relieve the pain, using chloral or cannabis indica, and not overlook the administration of such drugs as aromatic spirits of ammonia, tincture of ginger, tincture of capsicum, and follow with a reasonable dose of a saline physic. Generally saline physics are best for cattle and oils for horses, under comparable conditions.

#### DIARRHEA

I desire to direct to your attention the dairy-herd diarrhea which starts with one animal, and within a few days involves the entire herd. This condition is characterized particularly by the great amount of feces voided over night or during the day, each passage becoming thinner, until the stool becomes watery and is mixed with blood. It is accompanied by slight elevation of temperature, diminished milk flow, lessened appetite, dehydration and general emptiness. To me this is an intestinal hemorrhagic septicemia, and at its outset calls for the administration of hemorrhagic septicemia bacterin to all animals at once, and satisfactory results will follow.

Hemorrhagic septicemia organisms are usually present in the animal economy and in fact everywhere, and when they become pathogenic, cause trouble. This is true also of other organisms.

The colon bacillus is ever present in the intestinal tract, and occasionally it becomes pathogenic, and when it does it causes serious trouble, characterized by diarrhea, emaciation and death. No certain diagnosis can be made without using a laboratory, and no biological or pharmaceutical treatment avails much if any at all.

#### ACUTE BLOATING

Acute bloating of cattle is seen frequently and most common during pasturing in the early morning, during cool, moist weather in spring and in fall. The danger is further increased by watering immediately after pasturing or after feeding. Possibly in some cases there is a predisposition to bloating, varying according to the susceptibility of the individual. Greedy feeding and succulent feed, however, contribute much as factors. Some years are worse than others, and the same can be said of pastures.

Care should be exercised always not to turn cattle out on pasture until the grasses and clover have had an opportunity to dry. Even then, once the condition is known to exist in the herd or on the farm, it is deserving of anxiety on the part of the herd-owner, as no one can tell when it may happen. For my part I always left with the owner one or more sealed quart Mason fruit-jars, of pea-size granular hyposulfite of soda, with instructions to give four heaping tablespoonfuls in a pint of warm water every 15 or 20 minutes, after an initial dose of one-half a pound in a quart of water as a drench.

Formalin is also indicated and is harmless when administered properly diluted. Every known method to induce belching should be followed, the mouth should be kept open, and even a tickling or swabbing of the velum and fauces is indicated for the purpose intended.

The anterior portion of the body should never be depressed, but rather always elevated, and massage of the rumen can be only beneficial. If suffocation is threatening, artificial opening of the rumen or the use of the trocar is indicated at once.

#### CHRONIC BLOATING

To me I am inclined to think this is a symptom which may be present in a variety of diseases of the fore stomach, the abomasum, the esophagus or the intestines.

Atony of the rumen is a common cause, with which may be included traumatic gastritis, stenosis of the esophagus, and

stenosis of the intestine, not overlooking intestinal constipation and obstipation.

#### ATONY OF THE FORE STOMACHS

Atony of the fore stomachs consists in a slowly developing weakness of contraction of these organs, caused by a diminution of irritability and energy of contraction. The removal of food in normal quantity is retarded and thus digestion becomes disturbed.

The causes include over-exertion, prolonged excitement, lack of secretion, dilation of the rumen, and prolonged improper feeding of dry, non-digestible, non-nutritious feeds, such as wheat straw, chaff and marsh hay. In calves it develops subsequent to or with gastro-enteritis.

Predisposing causes are debility after chronic, acute and febrile diseases, and in advanced pregnancy all stomachs may become so compressed by the pregnant uterus that atony results.

In treating these cases rough feed should be of good quality and the quantity restricted greatly. Mashers mixed with common salt are indicated. Pasturing has a good dietetic effect. Drugs stimulating muscular movement of the rumen are useful and should be given for a long time.

Alcohol is beneficial, as it stimulates the movements of the rumen. Nux vomica and strychnine are always indicated. Purgatives should be withheld unless constipation is evident. Hydrochloric acid, one tablespoonful to a quart of water three times a day, is beneficial, and always water and more water is indicated.

With these, the more common diseases of the digestive tract of cattle, I will end this discourse, hoping you will have derived some degree of benefit comparable to the pleasure it has been to me in preparing and delivering it to you.

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#### Illinois Veterinarians to Meet in Springfield

Dr. W. B. Holmes, of Springfield, Ill., is chairman of the committee that will have charge of the local arrangements for the annual meeting of the Illinois State Veterinary Medical Association in that city on February 18 and 19. Associated with Dr. Holmes are Dr. C. C. Hastings, Williamsville; Dr. A. E. Dickerson, Springfield; Dr. E. E. Sweebe, North Chicago, and Dr. A. E. Bott, East Saint Louis. Headquarters will be at the Abraham Lincoln Hotel. A program of unusual entertainment is being planned for the evening of the 18th, for both gentlemen and ladies. A good literary program is promised.

# ABSTRACTS



THE RECOVERABILITY OF MYCOBACTERIUM TUBERCULOSIS AVIUM FROM EXPERIMENTALLY INFECTED GUINEA PIGS. William H. Feldman. Jour. Inf. Dis., lix (1936), p. 1.

Thirty-four guinea pigs were inoculated subcutaneously with suspensions of virulent *Mycobacterium tuberculosis*. Necropsy was made from one to 70 days subsequent to injection and the spleens were emulsified and cultured. The avian tubercle bacillus was obtained from eight of the 20 guinea pigs examined at necropsy up to and including 21 days after receiving the infective inoculum. Of the 14 animals examined at necropsy, 28 to 70 days after the infection was introduced, 13 yielded the specific microörganism. Macroscopic lesions of a tuberculous character were present in only one of the 43 animals examined. The experiments showed that the organism of avian tuberculosis may be recovered from the spleens of previously inoculated guinea pigs, although gross lesions of tuberculosis usually are absent. The author suggests the advisability of culturing the spleens of guinea pigs routinely in the study of clinical material in which the presence of the avian tubercle bacillus is suspected.

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AN IMPORTANT FACTOR IN THE MECHANISM OF SPECIFIC BACTERIAL AGGLUTINATION. C. R. Donham and C. P. Fitch. Jour. Inf. Dis., lix (1936), p. 7.

Water included in the antigen preparation is inhibitory of agglutination. The authors suggest that this adds one more complicating feature to the complex problem involving the measurement and control of the forces affecting the mechanism of agglutination. A practical method of recognizing this factor in the mechanism of agglutination and for overcoming its effect upon agglutination results used in the diagnosis of disease is not available. The agglutination method has been used in the diagnosis of disease and identification of bacterial species with an acceptable degree of accuracy. The experiments suggest the need for experiments designed to find a more suitable suspending

medium for bacterial agglutination antigens instead of the usual saline solutions.

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THE ALTERATION OF THE BACTERICIDAL POWER OF THE BLOOD IN THE PRESENCE OF INFLAMMATION. Howard W. Hughes. Brit. Jour. Exp. Path., xvii (1936), p. 369.

The bactericidal power of normal human blood does not fall *in vitro* within six hours. The blood of patients with active bacterial disease shows a steady loss of bactericidal power. Normal persons when given vaccine show a similar change during the stage of reaction. Blood drawn during a rigor shows one of two changes. If drawn at the beginning of rigor, a constant low level of bactericidal power without further loss on standing occurs. If it is drawn later, it shows a bactericidal power below normal and a progressive loss *in vitro*. Blood drawn after the rigor is normal. A mixture of serum taken from a patient with active bacterial disease or with an artificially produced reaction and normal blood-cells shows a constant low bactericidal power with no further deterioration.

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ANTIBACTERIAL IMMUNITY TO INFECTION WITH HEMOLYTIC STREPTOCOCCI IN RABBITS. T. C. Stamp. Brit. Jour. Exp. Path., xvii (1936), p. 391.

Immunity to infection with hemolytic streptococci believed to be mainly antibacterial may be developed in rabbits against intravenous and intradermal infection with the homologous strain. Immunity to intravenous infection probably is developed before that to intradermal infection. This immunity may be passively transferred to normal rabbits and it is not effective against infection with a heterologous strain.

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CENTRIFUGATION STUDIES. I. Critical examination of a new method as applied to the sedimentation of bacteria, bacteriophages and proteins. W. J. Elford. Brit. Jour. Exp. Path., xvii (1936), p. 399.

A new technic is described enabling the sedimentation of bacteria, bacteriophages, viruses and proteins to be studied with the ordinary bucket type of high-speed centrifuge. The density of *Bacillus prodigiosus* has been determined as 1.10 and that of *Staphylococcus K* bacteriophage as 1.25. Bacteriophages have densities approaching those of proteins, indicating a compact structure in contrast to the cellular morphology of bacteria which

have high specific water contents and correspondingly low densities, 1.05 to 1.15. The following particle sizes were determined: (1) *B. prodigiosus*, 0.7 to 0.8 microns; (2) *Staph. K* phage, 60 to 70 millimicrons; (3) antidysentery phage, 15 to 17 millimicrons; (4) hemocyanin, 22 millimicrons, and (5) edestin, 8 millimicrons.

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CENTRIFUGATION STUDIES. II. The viruses of vaccinia, influenza and Rous sarcoma. W. J. Elford and C. H. Andrews. Brit. Jour. Exp. Path., xvii (1936), p. 422.

The particle diameter of vaccinia virus has been determined by means of centrifugation to be 170 to 180 millimicrons, that of influenza virus 87 to 99 millimicrons and that of Rous sarcoma virus to be 60 to 70 millimicrons.

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STUDIES OF THE CLINICAL MANIFESTATIONS AND TRANSMISSIBILITY OF INFECTIOUS CORYZA OF CHICKENS. J. R. Beach and O. W. Schalm. Poultry Sci., xv (1936), p. 466.

Infectious coryza occurs as a simple coryza manifested only by a nasal discharge or as a coryza with complications consisting of edematous swelling of the face, sinusitis, conjunctivitis, tracheitis, bronchitis, and infection of the air-sacs. The disease is easily transmitted by inoculation of normal chickens with exudative material from any infected part or by direct contact exposure of healthy fowls to diseased ones. Transmission of the disease by indirect contact was accomplished but did not take place easily. Cages contaminated by occupancy, by diseased chickens and by having virulent exudate from infected chickens placed in the feed and water-vessels, did not remain infective to susceptible chickens placed in them for longer than 24 hours. The disease was transmitted by inoculating healthy chickens with scrapings of the nasal mucosa of chickens which had been infected with coryza but had shown no symptoms for as long as 46 days. Pigeons were found to be refractory to the disease. Three turkeys which were tested were found to be susceptible and the resultant disease was of the same character as natural cases of coryza and sinusitis in this species of bird.

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STUDIES OF INFECTIOUS CORYZA OF CHICKENS WITH SPECIAL REFERENCE TO ITS ETIOLOGY. O. W. Schalm and J. A. Beach. Poultry Sci., xv (1936), p. 473.

For the maintenance of slant or plate cultures of *Hemophilus gallinarum* it is necessary to transfer at intervals of less than

14 days. The optimum temperature for growth is from 34 to 42° C., minimum temperature 25° C. and maximum temperature 45° C. The injection of pure cultures of *H. gallinarum* into the respiratory tract of fowls produced a coryza with symptoms identical with those of the natural or exudate-induced disease but of much shorter duration. Experiments designed to determine whether the longer duration of the natural or the exudate-induced disease was due to the presence in the exudate of bacteria other than *H. gallinarum* or of a filtrable virus yielded negative results. A mild, culture-induced coryza of only two days duration by rapid serial passage through chickens increased in virulence and severity until it was of the same character as that produced by exudate obtained from severe field cases. This increase in virulence and severity occurred without the introduction of any pathogenic agent other than the culture of *H. gallinarum* with which the series of passages was initiated. The results are regarded as evidence that the mild character of the culture-induced disease is due to a decrease in virulence of the organism on artificial media and that the virulence can be restored by continued cultivation in the more favorable environment of the nasal passages of susceptible chickens.

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STUDIES ON THE SENSITIZATION OF ANIMALS WITH SIMPLE CHEMICAL COMPOUNDS. III. Anaphylaxis induced by arsphenamine. K. Landsteiner and John Jacobs. Jour. Exp. Med., lxiv (1936), p. 717.

Experiments are described which show that with a given treatment guinea pigs can be sensitized to arsphenamine so that a considerable percentage die in anaphylactic shock on intravenous administration of the substance.

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AN ACQUIRED RESISTANCE OF GROWING ANIMALS TO CERTAIN NEUROTROPIC VIRUSES IN THE ABSENCE OF HUMORAL ANTIBODIES OR PREVIOUS EXPOSURE TO INFECTION. Peter K. Olitsky, Albert B. Sabin and Herald R. Cox. Jour. Exp. Med., lxiv (1936), p. 723.

As mice grow older, they acquire a resistance to peripheral inoculation with the Indiana and New Jersey strains of vesicular stomatitis virus and to some extent also to western equine encephalomyelitis virus but little or none to the eastern strain. While some mice may become resistant as early as the 30th day of life, others may still be susceptible at one year of age. The resistance is readily demonstrable when the inoculations are made by intra-

nasal, subcutaneous, intramuscular, intraperitoneal and intravenous routes, but not when the virus is injected directly into the brain. The resistance is not related to previous exposure to infection or to the presence of specific or nonspecific antiviral bodies in the blood. No difference in susceptibility to peripheral inoculation was found in young and old guinea pigs to pseudorabies virus and in relatively young and old *Macacus rhesus* monkeys to poliomyelitis virus.

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THE STANDARDIZATION OF LONGEVITY AGAINST DOSE IN EXPERIMENTAL TUBERCULOSIS BY INTRACEREBRAL INOCULATION. Kenneth C. Smithburn. Jour. Exp. Med., lxiv (1936), p. 771.

The intracerebral inoculation of tubercle bacilli into normal guinea pigs induces acute meningoencephalitis with minor metastatic lesions. The disease is fatal in a relatively short time and is characterized by a rather typical succession of symptoms and a fairly characteristic temperature curve. The disease is produced by very small numbers of bacilli, and under standard conditions survival time is so uniform as to make possible quantitative or titration experiments.

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THE SUSCEPTIBILITY OF SWINE TO THE VIRUS OF HUMAN INFLUENZA. Richard E. Shope and Thomas Francis, Jr. Jour. Exp. Med., lxiv (1936), p. 791.

Swine inoculated intranasally with human influenza virus alone develop an ill-defined, mild and usually afebrile illness of short duration. The anterior lobes of the lungs of such animals on postmortem contain scant scattered areas of lobular atelectasis. Transmission of the virus for five serial passages through two groups of swine failed noticeably to enhance its pathogenicity for this species. The disease produced in swine by infection with human influenza virus alone is indistinguishable clinically and pathologically from that caused by infection with swine influenza virus alone.

Transmission of human influenza virus from swine to swine by contact succeeded in only one of four attempts. Swine inoculated intranasally with a mixture of human influenza virus and *H. influenza suis* usually develop a febrile, depressing illness similar to mild swine influenza. The pneumonia encountered in such animals at autopsy is similar to but less extensive than that seen in swine influenza. In some animals *H. influenza suis* failed to become established and the disease then seen is identical with that caused by human influenza virus alone. The human influenza

virus recovered after five serial transfers in swine was immunologically the same as that with which the experiments were begun.

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STUDIES ON HEMOLYTIC STREPTOCOCCI. III. *Streptococcus equi* and related strains. Alice C. Evans. Jour. Bac., xxxii (1936), p. 541.

A review of the literature on *Streptococcus equi* and the characters of the species are described as observed in 20 cultures isolated in various parts of the United States and Denmark (4 strains). Eleven strains agreed in every character; one of them was chosen as the type strain. Nine strains are regarded as variants because they differ from the type strains in one or more characters yet resemble *S. equi* more closely than any other species. In a collection of about 400 strains from human disease sources not one was classified as *S. equi*. The human strains which had been reported by other investigators to be *S. equi* were found to belong to other groups.

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THE INTERPLAY OF CELLS OF THE HEMATOPOIETIC TISSUE IN RABBITS INFECTED EXPERIMENTALLY WITH THE TUBERCLE BACILLUS. THE ORIGIN OF THE MONOCYTE CONSIDERED. E. M. Medlar and K. T. Sasano. Amer. Jour. Path., xii (1936), p. 825.

All systems of the hematopoietic tissues are involved during the process of acute tuberculosis infection in rabbits. The stem cells are greatly increased. Undifferentiated cells, perhaps stem cells, enter the circulation, retain the ability to multiply within the circulating blood and also in all probability after entering tissues other than blood-forming tissues. The cells in the germinal centers of the spleen are probably stem cells which in acute tuberculosis in the rabbit differentiate into monocytes; pulp cells in the spleen probably have the same origin.

Monocytes also originate from at least some portions of the lymphoid tissue outside the spleen. No evidence was found to indicate that the monocyte arises from the marrow or from the reticulo-endothelial system outside of the lymphoid system. The criteria on which the concept of a reticulo-endothelial system rests are insufficient to establish such a system. The circulating blood mirrors the change found in the so-called fixed hematopoietic tissues, although there may be a lag of several days before changes are reflected. The interplay of various cell types of the hematopoietic tissue necessitates a consideration of this tissue as a whole in pathological processes where it may play an important rôle.



### Regular Army

Captain Edgerton L. Watson is relieved from his present assignment and duty at San Francisco Port of Embarkation, Fort Mason, Calif., effective at such time as will enable him to sail on the transport scheduled to leave San Francisco, Calif., on or about March 12, 1937, for the Hawaiian Department, and upon arrival will report to the commanding general for assignment to duty with the Veterinary Corps.

Major Harry E. Van Tuyl is relieved from duty at Fort Hamilton, N. Y., effective at such time as will enable him to proceed to New York, N. Y., and sail on or about March 18, 1937, via Government transportation, for the Philippine Department, for duty.

Captain Ernest E. Hodgson is assigned to duty at Fort Ringgold, Texas, effective upon completion of his present tour of foreign service in the Hawaiian Department.

Major Frank C. Hershberger is assigned to duty at Fort Hamilton, N. Y., effective upon completion of his present tour of foreign service in the Philippine Department.

Each of the following-named officers of the Veterinary Corps is directed to proceed from Washington, D. C., to Carlisle Barracks, Pa., and report on December 30, 1936, to the Commandant, Medical Field Service School, for temporary duty for a period of approximately five months for the purpose of pursuing the basic course of instruction.

1st Lt. Velmer W. McGinnis

1st Lt. Lloyd C. Teske

### Veterinary Reserve Corps

#### NEW ACCEPTANCES

Leonhard, Ernest H.....1st Lt...RFD 2, Longmont, Colo.

Lindsey, Mark Bay.....1st Lt...7102 Rita Ave., Huntington Park, Calif.

#### PROMOTIONS

##### To

Cavanaugh, Jos. L.....1st Lt...R. 1, Esbon, Kan.

Jewell, Harold J.....1st Lt...116 N. W. 7th St., Ardmore, Okla.

#### NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Werrin, Milton .....1st Lt...Hq. 3rd Corps Area, Baltimore, Md.

#### TERMINATION OF ASSIGNMENT TO ACTIVE DUTY

Thiele, Arthur R.....1st Lt...Fort Bragg, N. C.

Watkins, Ernest St. J.....1st Lt...Medford, Ore.

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# COMMUNICATION

## CORRECTION OF MISINFORMATION ON EMPLOYMENT OF PER DIEM VETERINARIANS

TO THE EDITOR:

In the October, 1936 issue of the JOURNAL of the A. V. M. A., which contains the proceedings of the Columbus meeting of the House of Representatives of that Association, several inaccurate statements appear which I wish to correct in order that veterinary history in this country may be kept straight.

For instance, on page 484, it is reported that Dr. D. B. Palmer, of Minnesota, made a request (please note that this was on the evening of August 13, 1936) to memorialize the Bureau of Animal Industry:

\*\*\* to continue its employment of practicing veterinarians on a per diem basis. As I understand the situation now, most men, at least those who are under 45 years of age, are going to be required, if they are going to do any more field work for the Bureau of Animal Industry, to take the civil service examination. \*\*\* They are going to be laid off. Some have already been dismissed.

Whereupon, he moved, among other things:

\*\*\* that a sufficient and trained personnel of veterinary practitioners be maintained in communities where this work is under way \*\*\* that the per diem employment of practicing veterinarians in this program be continued as it was prior to July 1, 1936, and that mileage and subsistence be allowed when it is required that they be away from their official stations, \*\*\*

In discussing his motion, Palmer states, on page 485:

In the first place it will remove everybody who is over 45 years of age. \*\*\* Under the old plan, we worked in our own districts. We are closed out of that now, and we must either accept one of these jobs where they can send you here, there, and yon, or be out of the picture. \*\*\* We have not yet found out why it was issued in the first place. We have received many letters, none of which answered the question as to why it was necessary to make the ruling. \*\*\* We have a letter from the President of the United States Civil Service Commission in which he states, over his signature, that any plan which is suitable to the Bureau of Animal Industry will be satisfactory to the Civil Service Commission. We have asked the Bureau of Animal Industry repeatedly about it. I am very sorry Dr. Mohler is not here in person to tell us why, but the point is we have never received an answer.

Secretary Hoskins then entered the discussion and gave an accurate statement of the reason for the Civil Service Commis-

sion entering into the question of employing per diem veterinarians after June 30, 1936. This was along the lines of the information I gave to Dr. Hoskins in his Chicago office in June, 1936. In addition, this same information was furnished Dr. C. P. Fitch in my letter of April 13, 1936, referred to below. Dr. Palmer is quoted as saying:

We have asked the Bureau of Animal Industry repeatedly about it. \* \* \* but the point is we have never received an answer.

I do not know who his "we" refers to, as this Bureau has never received any letter from Dr. Palmer on the subject and, of course, this being the case, he could not expect to obtain a reply. On the other hand, if "we" refers to the officials of the veterinary association of his state, is it fair to say "we have never received an answer" in view of the following documentary evidence? The first letter we received on this subject was dated April 2 and was signed by R. A. Merrill, president, and C. P. Fitch, secretary, of the Minnesota State Veterinary Medical Society. I made reply by air mail on April 4, explaining the apparent misunderstanding of the writers and stated that the Solicitor of the Department had been asked for an opinion as to whether it would still be possible to make appointments of veterinarians after June 30, 1936, without reference to civil service rules and regulations. Dr. Fitch acknowledged this letter on April 11, thanked me and asked: "Have you any information as to why we have had no reply from the U. S. Civil Service Commission?" My answer, dated April 13, 1936, to Dr. Fitch, stated that the Solicitor had expressed his belief:

that the language in the Appropriation Act in the form in which it has been enacted requires employment in accordance with civil service rules and regulations.

On April 22, Dr. Fitch again thanked me for the above letter and asked to be kept informed as to the plan which might be worked out "to employ practicing veterinarians on a part-time or cooperative basis per annum."

Again, on May 26, 1936, Dr. Fitch wrote asking:

Would it be possible for you to let us know the present status of this matter?

My reply of May 29 stated:

This subject has been submitted to the U. S. Civil Service Commission and negotiations are still in progress. Until the Commission's approval is obtained, we are unable to give you any assurances in the premises.

On July 1, 1936, I wrote to Dr. Fitch as follows:

I am pleased to state that arrangements have now been completed with the U. S. Civil Service Commission for the continued employ-

ment of the necessary number of full-time veterinarians pending the certification of civil service eligibles, as well as for the employment of local part-time veterinarians, so that there will be no impairment to the progress of the work. A copy of this letter is being sent to Dr. R. A. Merrill at Clara City for his information.

On July 6, Dr. Fitch acknowledged the receipt of this letter and stated:

It will be brought to the attention of our State Veterinary Society at its meeting to be held at University Farm, July 9 and 10, 1936.

It is assumed that this was done although I have no knowledge of Dr. Palmer's presence at Saint Paul. However, I am of the opinion that had the latter showed as much interest previous to the A. V. M. A. meeting as he did on August 13, he could have received an accurate statement of the facts from the officials of his State Association. At the very time (August 13) he made his complaint there were 1,023 per diem veterinarians on the Bureau rolls, 169 of whom were in his state and 155 in Wisconsin. Since then the total number has been increased to 1,313, with 170 in Minnesota and 158 in Wisconsin. These are facts and not surmises and make one wonder where he received his information that

they are going to be laid off. \* \* \* (and will) be out of the picture.

Letters similar to those received from the officials of the Minnesota State Veterinary Medical Society were received from several other state veterinary organizations, but their source was identical. A rather amusing corollary resulted from the above activity. Within a short time, the Bureau received complaints from the graduating classes of a number of veterinary colleges and student chapters of the A. V. M. A., in which they registered their wish

that the Bureau employes continue to enjoy the privileges and protection of civil service examination. \* \* \*

and objected to our

intention to take over the large number of temporary veterinary inspectors into the regular B. A. I. force without passing the civil service examinations \* \* \* which would place considerable numbers of veterinarians who are graduating this year \* \* \* at a great disadvantage and materially reduce their chances of receiving a B. A. I. appointment after taking the civil service examination. We believe that they (the temporary veterinarians) would concur in our attitude if our side of this matter was brought to their attention.

Thus the Bureau was placed between two fires as a result of misinformation and misunderstanding.

In this connection it is my duty to challenge Dr. Palmer's statement when he claims

We have a letter from the President of the U. S. Civil Service Commission in which he states, over his signature, that any plan which is suitable to the Bureau of Animal Industry will be satisfactory to the Civil Service Commission.

Several letters have been received from the field signed by the President and others of the Civil Service Commission, but I have failed to note any such statement. I now challenge Dr. Palmer to produce that letter or a copy of it.

In order to have a full understanding of this entire subject it is necessary to go back to March, 1936. When it became likely that the funds allotted to us by the AAA, for Bang's disease and tuberculosis eradication work, would terminate on June 30, 1936, and that the regular appropriations for the present fiscal year would provide the funds for such work, we asked the Solicitor for an opinion as to whether this change in the source of funds would eliminate our authority for employing persons outside of civil service and require that after June 30 such appointees be in accordance with civil service rules. In his reply he stated:

Had this paragraph stopped after the phrase—"and for other purposes, as authorized by said section 37," there would be no doubt that appointments under this item could be made under the provisions of Section 10(a) of the Agricultural Adjustment Act, as amended. However, the inclusion in the paragraph of items which are in the Agricultural Adjustment Act and others which are not, such as the item for the purchase, maintenance, operation, and repair of passenger-carrying vehicles necessary in the conduct of field work outside the District of Columbia, is indicative that it was not the intent of Congress that the sums appropriated were to be used for the employment of personnel under the provisions of the Agricultural Adjustment Act, as amended, but in accordance with civil service rules and regulations.

This matter was then brought to the attention of the Civil Service Commission, which was asked for permission to allow us to continue under appointment, pending certification, those full-time veterinarians employed outside civil service and for permission for the appointment of local veterinarians on a per diem basis for intermittent and part-time duty.

The Commission replied after analyzing the situation, specifically having in mind the new fund in the current appropriation act, as follows:

It will be observed that there is no affirmative exemption from either the Civil Service Act and Rules or the Classification Act of 1923, as amended. Consequently in accordance with a long line of opinions of the Attorney General and Comptroller General's Decision, these statutes are for application as general law. So far as the Classification Act of 1923 is concerned, the compensation sched-

ules are for application by the Department in the same manner that it administratively allocates field positions. The Civil Service Act and Rules are, however, for full application under the jurisdiction of the Commission. This finding conforms to the opinion of the Solicitor of your Department rendered on April 9, 1936, to the Chief of the Bureau of Animal Industry.

It was determined that if the Agricultural Appropriation Bill became law in its present form, the problem facing the Department could be solved through the following procedure:

With respect to the junior veterinarians employed for part-time and intermittent service, the Department of Agriculture and the Civil Service Commission should join in recommending to President Roosevelt an amendment to Schedule A of the Civil Service Rules to be known as paragraph 7 of Subdivision IX, and to read as follows:

Any local veterinarian employed on a fee basis or a part-time basis, where in the opinion of the Commission the establishment of registers is impracticable.

This gives the Bureau the same privileges for part-time or fee-basis appointments as are granted to the U. S. Public Health Service and the U. S. Veterans' Administration and such employees on a per diem basis are entitled to mileage for personally owned automobiles under the same conditions as regular full-time Bureau employees.

We agreed to the Commission's recommendation regarding the employment of part-time local veterinarians and pressed our recommendation for the appointment, pending certification, of employees already in the service outside civil service.

The Commission asked that representatives of the Department discuss the matter with them, and on May 18, three Department representatives (unfortunately none of them was present at the Columbus, Ohio, meeting) had a conference with the Commission's representatives, with the result that the Commission granted authority for the continuation of such employees pending certification. At this conference it was made plain by the Commission's representatives that they were willing to request an executive order providing for the employment of local part-time or fee-basis veterinarians, with the understanding that they would be practicing veterinarians, employed locally from time to time as their practices permitted and their services were needed, and whose aggregate salaries would be held down accordingly. This Executive Order was issued as above quoted on June 23, 1936.

Not to my surprise, the Commission was most insistent that full-time veterinarians, whether on per annum or per diem basis,

should be employed only in accordance with Civil Service rules and after regular examination and certification.

Personally, I believe that the Civil Service Commission accorded the Bureau very sympathetic coöperation and went out of its way to be helpful in a very annoying situation. In fact, I must admit that the concessions we obtained from the Commission exceeded my fondest hope.

Before closing I cannot refrain from correcting another inaccurate statement made at the same meeting, which appears on page 415, as follows:

I know the federal government is so anxious to get our graduates that they will not even let them ripen. They take them right in the midst of the term.

I always feel charitable and tolerant regarding loose statements made in the heat of debate, but nevertheless the above quotation must be branded as erroneous. It is true that members of the graduating classes of approved veterinary colleges have been permitted to take the civil service examination for junior veterinarians before finishing their final year's work. However, although they may pass the examination successfully, they have never received appointment in the Bureau until after they have been graduated and until the fact of their graduation has been attested by the deans of the veterinary colleges concerned.

Very truly yours,

J. R. MOHLER, *Chief of Bureau*

*Washington, D. C., December 18, 1936.*

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### Adrenal Gland Function

An important function of the cortex of the adrenal glands is to regulate the amount of potassium in the blood and thus keep the body from poisoning itself, according to Drs. Raymond L. Zwemer and Richard Truszkowski, of Columbia University College of Physicians and Surgeons.

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### Consumer Meat-Grading

Rules and regulations covering grading and stamping of meats and meat products have been announced by the Bureau of Agricultural Economics, U. S. Department of Agriculture. The service has been expanded greatly since 1926, when the first rules and regulations were issued. It now includes the grading and stamping of beef, pork, lamb, mutton, and sausage products. The meat-grading and stamping service is voluntary. It is paid for by the applicants for such a service.



## SOUTHERN STATES VETERINARY MEDICAL ASSOCIATION

The twenty-first annual meeting of the Southern States Veterinary Medical Association was held at the Vanderbilt Hotel, Asheville, N. C., October 21-23, 1936. The meeting this year was held three weeks earlier than usual, so that those who do not get to see the Blue Ridge Mountains very often, might see them when their colors are at their best. This meeting also was the first one held since the Association voted to do away with annual dues and substitute a registration fee. In an organization of this kind, this seemed to be the wisest thing to do in order to lift the load from a few faithful members and distribute it among those who actually derive the benefits of attending each meeting.

The Committee on Local Arrangements planned the program almost entirely, and everyone who was listed to contribute was there to take his place. Dr. M. M. Leonard, chairman of the Committee, was called away from Asheville the day before the meeting to attend the funeral of his father. Mrs. Leonard and the other members of the Committee functioned so well that every part of the program went off smoothly. Col. Robert J. Foster, president of the American Veterinary Medical Association, attended this meeting, the first since he had been in office. He addressed the members on "The Army Veterinary Service and the American Veterinary Medical Association." He was also called upon for a few remarks at the banquet.

Others who contributed to the program and their subjects are as follows:

- "Conditions Met by the Large-Animal Practitioner," Dr. W. L. Boyd, Saint Paul, Minn.
- "Practical Uses of Pregnancy Tests in Mares," Dr. C. E. Salsbery, Kansas City, Mo.
- "The Veterinarian and Meat Inspection," Dr. D. R. Gillies, U. S. Bureau of Animal Industry, Washington, D. C.
- "The Value of Dextrose in Veterinary Medicine," Dr. Otto Stader, Ardmore, Pa.
- "Milk Hygiene," Dr. John G. Hardenbergh, Plainsboro, N. J.
- "Anesthesia," Dr. E. E. Sweebe, North Chicago, Ill.

"How to Make Money Out of the Mule," Dr. W. L. Stroup, Corinth, Miss.

"Practical Methods for the Prevention and Treatment of Canine Distemper," Dr. Adolph Eichhorn, Pearl River, N. Y.

"Animal Hospitalization," Dr. J. V. Lacroix, Evanston, Ill.

"Broadening the Field of the Veterinarian," Dr. A. H. Quin, Jr., Des Moines, Iowa.

"Trends in Swine Practice," Dr. Ashe Lockhart, Kansas City, Mo.

The evening of the first day was spent at an informal harvest jamboree. This included dancing and other stunts that were full of fun and the evening passed all too quickly.

The banquet was held the evening of the second day, with Dr. Don Kitchen, of Greenville, S. C., presiding as toastmaster. Judge Julius G. Adams gave the address of welcome. Dr. Kitchen then called on a number of prominent veterinarians for a few remarks. He then introduced the speaker of the evening, Honorable Robert R. Reynolds, United States Senator from North Carolina, who entertained the banquet guests for more than an hour.

The entire second day was devoted to a clinic. Dr. Stroup demonstrated casting and the use of various knots. Dr. Boyd directed the operations on large animals, and Dr. Lacroix was the chief surgeon in the section for small animals.

The ladies were kept busy with parties and sightseeing trips. By the end of the third day, everyone was satisfied that the local veterinarians and their wives had spared no effort in making the Asheville meeting a most pleasant and profitable experience.

The meeting next year will be held in Atlanta. Dr. M. M. Leonard, of Asheville, N. C., was elected president; Dr. J. F. Kagey, of Kingsport, Tenn., first vice-president; Dr. George L. Houchins, of Martinsville, Va., second vice-president, and Dr. M. R. Blackstock (reëlected), Spartanburg, S. C., secretary-treasurer.

M. R. BLACKSTOCK, *Secretary*

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## MARYLAND STATE VETERINARY MEDICAL ASSOCIATION

The summer meeting of the Maryland State Veterinary Medical Association was held October 28-29, 1936, at College Park. The sessions were well attended and proved both interesting and instructive. Dr. T. A. Ladson, of Olney, presided and briefly discussed the present status of the practitioner in relation to his clients and to the official forces in the state.

Dr. Harry W. Schoening, Chief of the Pathological Division, U. S. Bureau of Animal Industry, presented a most interesting

paper on "Swine Erysipelas." He reviewed our present knowledge of the disease and discussed the methods of diagnosis and treatment. Dr. Schoening pointed out that this disease is becoming an increasingly important factor in swine production in many sections of the United States and had been found to exist in certain parts of Maryland.

Dr. R. V. Smith, of Frederick, discussed several cases of *Br. abortus* infection in horses that have occurred in his practice. He reported that in virtually all instances where infection had occurred in horses the cattle on the premises showed a high percentage of Bang's disease reactors. The report of Dr. E. B.



MARYLAND VETERINARIANS AT COLLEGE PARK

Simonds, Baltimore, delegate to the House of Representatives of the American Veterinary Medical Association, indicated that the problems of the national organization are being carefully studied and seriously investigated.

Dr. E. F. Schroeder, of the Angell Memorial Hospital, Boston, Mass., presented a most interesting illustrated description of fractures in small animals. The small-animal clinic in the afternoon also was under the direction of Dr. Schroeder and was most practical, interesting and instructive. In the early evening an out-door get-together dinner was served at the laboratory of the Live Stock Sanitary Service. Barbecue beef and baked oysters with all the trimmings proved to be a popular change from formal banquets.

At the morning session on Thursday, October 29, Dr. A. L. Brueckner, Chief of the Live Stock Sanitary Service Laboratory, discussed biological tests for pregnancy. He illustrated the changes involved with several autopsied rats that had previously been injected with equine serums. Dr. W. T. Miller, of the Beltsville Research Center, discussed the several types of mastitis and various tests employed to detect them, together with the treatment of infected animals. Large sections of infected udders were used to illustrate the progressive changes due to mastitis infection.

The integration of profitable pasture grasses with the field of veterinary medicine was interestingly presented by Prof. J. E. Metzger, of the University of Maryland. It was indicated that the fertilization and proper management of the various pasture grasses play an important part in the nutrition and economical development of our various types of live stock.

Several demonstrations of the diagnosing of animal diseases were shown by Drs. L. J. Poelma and W. R. Teeter. Drs. H. M. DeVolt and C. R. Davis demonstrated a variety of poultry diseases with prepared specimens and recently autopsied birds. The large-animal surgery work was ably demonstrated by Majors James R. Sperry and James A. McCallam, Veterinary Corps, United States Army.

The famous "Gypsy Queen" reached the end of the longest trail ever traveled by a horse under saddle at the meeting. Between April 1, 1925, and November 4, 1927, this horse, ridden by Sergeant Frank M. Heath, hit every state in the Union and traveled 11,532 miles. Old age was taking its toll, however, so Sergeant Heath regretfully permitted her destruction and her skeleton will be used for instructional purposes at the University of Maryland.

MARK WELSH, *Secretary*

### VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The November meeting of the Veterinary Medical Association of New York City was held at the Hotel New Yorker, November 4, 1936.

At this meeting we had the pleasure of hearing from one of the country's outstanding authorities on nutrition, Dr. G. R. Cowgill, of the Department of Bio-Chemistry and Nutrition, School of Medicine, Yale University. The topic of his interesting discussion was "What Shall I Feed My Dog?" including a

scientific study of canine nutrition. In presenting this subject, Dr. Cowgill illustrated it with film and slides showing the value of vitamins B1 and B2 or G. A deficiency of these vitamins produces an ataxia, pellegra, polyneuritis, or complete collapse in the body structure. By means of charts the value and needs of vitamins, minerals, proteins, fats and carbohydrates were explained in reference to the importance in the maintenance of body functions.

Joining with Dr. Cowgill in the discussion of this topic was Dr. R. W. Russell, of the Department of Bio-Chemistry and Nutrition, Rutgers University, New Brunswick, N. J. This program was made possible through the efforts of Dr. M. L. Morris, who opened the evening's discussion by reading a paper outlining plans for the development of a council to set up standards whereby the nutritional value of dog foods could be judged, for the betterment of the dog and for the good of the dog-food industry.

The entire program was heartily received by an overflow attendance and the material presented in the different talks was highly profitable to all in attendance. A rising vote of thanks was tendered Drs. Cowgill and Russell for their addresses and to Dr. Morris for his paper and also for arranging this interesting program.

R. S. MACKELLAR, JR., *Secretary*

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### WESTERN MICHIGAN VETERINARY MEDICAL ASSOCIATION

The seventh annual meeting of the Western Michigan Veterinary Medical Association was held at the hospital of Dr. J. Y. Veenstra, on Kalamazoo Road, Grand Rapids, on December 10, 1936.

There was considerable discussion concerning veterinary legislation and the possibility of politics entering into the appointment of a new State Veterinarian. In the President's address Dr. J. Wm. G. Hansen, of Greenville, pointed out the importance of a united effort by all branches of veterinary service in order to bring about a greater efficiency.

Dr. H. H. Clark, of Lansing, gave a paper on the subject, "Diseases and Peculiarities of Cats." He gave special attention to the management of cats, as well as many personal experiences in handling the feline tribe.

Dr. Chas. Haasjes, of Shelby, gave a five-minute radio talk over station WASH, in Grand Rapids, at 1:30 P. M. He emphasized

the fact that the veterinary profession is instrumental in helping to sustain the life of the human family, that veterinary meetings are held for the benefit of the live stock industry, and that the complexity of animal diseases demands a broad knowledge of diseases in general for any one who attempts diagnoses.

Dr. B. A. Perry, of Hastings, discussed equine practice as we find it today. Dr. Herman Dykema, of Muskegon, gave an interesting talk on "The Diet of Small Animals."

Dr. Chas. Haasjes, as chairman of the Committee on Socialized Veterinary Medicine, of the Michigan State Veterinary Medical Association, made a preliminary report. He stated that the problem must be studied to learn whether this form of service will be better for the community than the service rendered by private practitioners to individual clients.

Dr. A. E. Erickson, of Charlotte, exhibited several reels of films taken in his practice. It took an hour to show these films. The doctor has been able to photograph various activities of the general practitioner as he finds them in his everyday practice.

Dr. E. C. W. Schubel, of Blissfield, secretary of the Michigan State Veterinary Medical Association, was a welcome visitor. He took an active part in the various discussions. He was given a vote of thanks for his efforts in helping improve conditions for the profession as well as assisting in the meeting. President Hansen conducted the question-box.

Dr. Robert Veenstra and Dr. H. H. Clark were selected as a committee to assist in conducting some laboratory demonstrations at the clinic to be held next summer. It was voted to hold the next annual meeting at Greenville, where Dr. Hansen would act as host.

The following officers were elected for the ensuing year: President, Dr. Herman Dykema, Muskegon; vice-president, Dr. H. H. Clark, Lansing, and secretary-treasurer, Dr. Chas. Haasjes (reëlected), Shelby.

CHARLES HAASJES, *Secretary*

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### SOUTH DAKOTA VETERINARY MEDICAL ASSOCIATION

The 17th annual meeting of the South Dakota Veterinary Medical Association was held at the Cataract Hotel, Sioux Falls, December 10-11, 1936. The president, Dr. A. A. Fosterman, of Utica, introduced Mr. Paul K. Myers, secretary of the Sioux Falls Chamber of Commerce, who extended a very hearty welcome to the veterinarians in attendance and asked them to make use of

the services offered by the Chamber of Commerce to make the meeting a most successful and enjoyable one. President Fosterman responded to the address of welcome.

Following the reading and approval of the minutes of the previous meeting, Dr. F. E. Walsh, of Iowa State College, Ames, Iowa, gave a talk on "Veterinary Practice." This was very much enjoyed and a good discussion followed.

Dr. I. J. Kleaveland, of Sioux Rapids, Iowa, gave a very instructive paper on "Cattle Practice," along with some case reports which brought out a very enthusiastic discussion, especially those dealing with cane and cornstalk poisoning, conditions which have been the source of constant trouble to practicing veterinarians recently. Following the discussion of Dr. Kleaveland's paper, a round-table discussion on sheep diseases took place. Scabies of sheep and the proper handling of an outbreak are subjects of considerable importance to all veterinarians, due to the fact that cases of scabies have been encountered in South Dakota during the past year.

Col. Robert J. Foster, president of the American Veterinary Medical Association, gave a very interesting talk on the national organization and the benefits to be derived from membership in it. He outlined plans which are being considered for making the JOURNAL more practical and useful for all branches of the veterinary profession. His talk was very well received and he may feel assured that the large group of veterinarians who heard him now have a clearer understanding of the workings of the A. V. M. A.

Over 100 veterinarians and their wives and friends attended the banquet Thursday evening. President Fosterman acted as toastmaster and presented Dr. Walsh, Col. Foster and Dr. Adolph Eichhorn for short talks. Dr. Eichhorn gave a very interesting account of his recent trip to South America, with particular reference to veterinary activities and the live stock industry in Argentina.

The first number on the program the morning of the second day was a talk by Dr. Eichhorn on recent developments and investigations in connection with virus diseases. This talk brought out a lot of questions, which resulted in a very enlightening discussion of this very important subject.

Dr. J. B. Taylor, of South Dakota State College, Brookings, gave a talk on poultry diseases that are found in this area. This proved to be very interesting, as Dr. Taylor presented both the laboratory and the practical sides of cases which he reported.

"Selenium Poisoning," presented by Mr. Alvin Moxon, chemist of South Dakota State College, was very interesting, as he has

made an extensive study of this subject and had pictures of specimens with which he illustrated his talk.

Dr. T. H. Ruth, Director of the South Dakota Division of Animal Industry, reported on the legislation which will be introduced at the next session of the legislature in January. This has been prepared by the South Dakota Live Stock Association, and the subject created quite a lively discussion.

Dr. C. H. Hays, of Pierre, gave a very able report on the work being done by the U. S. Bureau of Animal Industry in coöperation with state forces, with reference to tuberculosis eradication and Bang's disease control since the work was started November 28, 1934.

Resolutions were adopted covering the deaths of three South Dakota veterinarians, Drs. J. P. Niederauer, Thomas Roberts and C. D. Tuttle. Another resolution complimented the Associated Serum Producers, Inc., for the splendid publicity given and favors shown the veterinary profession in their radio broadcasts and advertising.

The election of officers resulted as follows: President, Dr. H. D. Perry, Yankton; vice-president, Dr. F. F. Smith, Emery; secretary-treasurer, Dr. G. E. Melody (reëlected), Gettysburg; delegate to A. V. M. A. House of Representatives, Dr. G. E. Melody, and alternate, Dr. A. A. Fosterman.

There were 115 veterinarians registered at this meeting, a very large attendance considering the fact that a large number engaged in federal-state work were unable to attend.

G. E. MELODY, *Secretary*

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## VERMONT VETERINARY MEDICAL ASSOCIATION

The twenty-ninth annual meeting of the Vermont Veterinary Medical Association was held at the Montpelier Tavern, Montpelier, December 15, 1936. Dr. H. L. Mills, of Burlington, presided and gave a very interesting presidential address. Reports of the officers were read and accepted.

Vermont had been declared a modified accredited area by the U. S. Department of Agriculture on November 2, so this meeting was held in conjunction with Vermont Achievement Day. Veterinarians were present from a number of the other New England states. No papers were read, although several business matters were discussed.

Officers for the new year were elected as follows: President, Dr. Henry C. Stetson, Saint Johnsbury; first vice-president, Dr. David A. Hopkins, Brattleboro; second vice-president, Dr. William

Philipsen, Brandon; secretary-treasurer, Dr. G. N. Welch (re-elected), Northfield, and representative to the A. V. M. A. House of Representatives, Dr. L. H. Adams (re-elected), Montpelier.

The Achievement Day program began at 2:00 o'clock in the Hall of Representatives at the State House. Hon. Edward H. Jones, Commissioner of Agriculture, presided and the welcome to Vermont was given by Hon. George D. Aiken, Lieutenant-Governor. The welcome to Montpelier was given by the Mayor, Hon. W. H. Dyer. Then officials of the other New England states exchanged greetings and offered congratulations to the Vermont officials. Dr. L. H. Adams, B. A. I. inspector-in-charge of tuberculosis eradication in Vermont, then responded in his usual pleasing manner.

Hon. E. S. Brigham, vice-president of the National Life Insurance Company, and former Commissioner of Agriculture of Vermont, reviewed the progress of tuberculosis eradication during the past 15 years. Dr. A. E. Wight, Chief, Tuberculosis Eradication Division, U. S. Bureau of Animal Industry, reviewed the work from the standpoint of a federal official.

At 6:30 o'clock, the meeting adjourned and everybody proceeded to the City Hall, where a cabaret banquet and a lively floor show were enjoyed by 335 persons. Dancing followed until the wee small hours of the morning.

G. N. WELCH, *Secretary*

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### Ten Texas Counties Released from Quarantine

Ten counties in Texas were released from the fever-tick quarantine on December 1, 1936. This is the largest area released since 1932, when 13 counties were released.

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### New Strain of Yellow Fever Virus

A new strain of yellow fever virus has been isolated by scientists of the International Health Division of the Rockefeller Foundation, promising more effective vaccination against yellow fever. A new hazard from yellow fever was discovered when the scientists found that Brazilian jungle fever is a yellow fever and that other mosquitoes besides *Aedes aegypti* can carry the disease.

# NECROLOGY



## TOM CARPENTER

Dr. Tom Carpenter, of Alameda, Calif., died of a heart attack, November 17, 1936, in the Alameda Sanatorium.

Born in Dartmouth, Devonshire, England, June 19, 1862, he accompanied his parents to America and settled with them in California when he was only seven years old. He attended public schools in San Francisco and then returned to England where he attended the Exeter Preparatory School for one year. He then entered the Ontario Veterinary College and was graduated in 1888.

The position of sanitary inspector was created for Dr. Carpenter shortly after he located in Alameda. He assumed this position on June 1, 1894, when the city was still in its infancy. He pointed out the dangers of bovine tuberculosis, and he is credited with having been the first veterinarian to employ the tuberculin test in California. One of the local newspapers said this of Dr. Carpenter:

His passing marked the close of a long and brilliant medical and research career that won him nation-wide acclaim and recognition. \* \* \* The fact that Alameda down through the years has been practically free from epidemics shows that Dr. Carpenter did his job well.

Dr. Carpenter joined the A. V. M. A. in 1926. He was a member of the Bay Counties Veterinary Medical Association and the Twelfth International Veterinary Congress. He was a charter member of the Alameda Kiwanis Club, and for thirteen consecutive years was grand marshal for the Club's annual Hallowe'en parade for the youngsters of the city. He led the parade three years ago. His fraternal affiliations were numerous and included the following: Oakland Lodge, No. 188, F. & A. M.; Alameda Chapter, No. 70, Royal Arch Masons; Alameda Commandery No. 58, Knights Templar; Aahmes Temple, Shrine; Alameda Lodge, No. 1015, B. P. O. E.; Derby Lodge, No. 258, Sons of Saint George; Alameda Lodge, No. 132, W. O. W., and Alameda Aerie, No. 1076, F. O. E., of which he was past state president. He is survived by his widow (née Helen Martin), one daughter and three grandchildren.

**GERRET EARLE MIDDLEHOFF**

Dr. G. E. Middlehoff, of Oroville, Calif., died at his home, March 16, 1936. He had been failing in health for some time and had been confined to his bed for four months prior to his death.

Born in San Francisco, Calif., March 5, 1882, Dr. Middlehoff attended public schools in Berkeley, and was graduated from the San Francisco Veterinary College with the class of 1911. He was a member of one of the oldest San Francisco families. Both of his grandfathers were pioneers of the gold rush days of 1849.

For a time after graduation, he was employed in a veterinary hospital. He then entered the service of the U. S. Bureau of Animal Industry and was assigned to meat inspection at Omaha. Later he was transferred to San Francisco. In 1916, he resigned from the Bureau and moved to Oroville, where he purchased an orange orchard and also engaged in general practice.

Dr. Middlehoff joined the A. V. M. A. in 1928. He was a member of the California State Veterinary Medical Association, and had been secretary of the California State Board of Examiners in Veterinary Medicine since 1926. He was County Live Stock Inspector, manager of the Butte County Citrus Association, and assessor and tax collector of the Thermalito Irrigation District. From 1917 to 1921, he was president of the Oroville National Farm Loan Association, and in 1932, he was chairman of the Butte County Work Relief Committee. He was a member of the Fellows Club, a charter member of the Oroville Lodge of Elks, a member of the Native Sons of the Golden West, a 32nd degree Mason and past commander of the Oroville Commandery No. 5 of the Knights Templar.

Surviving Dr. Middlehoff are his widow (née Laurie Lee) and a sister. Interment was in the Mountain View Cemetery, Oakland.

C. U. D.

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**HARRY WINTER PAXTON**

Dr. Harry W. Paxton, of Sioux City, Iowa, died at his home on July 5, 1936. He was a graduate of Iowa State College, class of 1914. He was in the service of the U. S. Bureau of Animal Industry, from July 23, 1934, until February 13, 1936, on emergency work in connection with tuberculosis eradication and Bang's disease control. He is survived by his widow and his mother.

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**E. MAKINS, JR.**

Dr. E. Makins, Jr., of Abilene, Kans., died on July 16, 1936, a victim of heat prostration. He was a graduate of the Kansas City Veterinary College, class of 1900, and had been in general practice at Abilene ever since his graduation. He was a member of the A. V. M. A. from 1902 until 1934, and was also a member of the Kansas Veterinary Medical Association.

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**HIRAM W. HAWLEY**

Dr. Hiram W. Hawley, of Chula Vista, Calif., died at his home, very unexpectedly, from a heart attack, September 9, 1936.

Born at Shopiere, Wis., March 22, 1862, Dr. Hawley attended local schools before entering the Chicago Veterinary College. He was graduated in 1891 and on November 1, 1895, entered the service of the U. S. Bureau of Animal Industry. He served at National Stock Yards and Chicago, Ill., until February 13, 1907, when he resigned. In July of the same year, he was reinstated and filled various assignments until his retirement on July 31, 1927.

Dr. Hawley joined the A. V. M. A. in 1918 and resigned shortly after he was retired from the B. A. I. service. He is survived by his widow and one brother.

T. A. S.

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**R. E. LARIMER**

Dr. R. E. Larimer, of Madrid, Iowa, died suddenly on October 6, 1936. He was a graduate of the Chicago Veterinary College, class of 1906, and had been in practice at Madrid ever since his graduation. He was a member of the Iowa Veterinary Medical Association.

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**WICKLIFFE A. PARKER**

Dr. W. A. Parker, of Eureka, Mo., died at Monroe, La., October 27, 1936. He had been in the service of the U. S. Bureau of Animal Industry, on emergency work, since July 9, 1934. He was a graduate of the Kansas City Veterinary College, class of 1912, and practiced at Eureka, Mo., until several years ago, when he accepted a position with the Board of Health of Saint Louis, Mo. He leaves his widow, one daughter and three sons.

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**FRANK W. HUEBEN**

Dr. Frank W. Hueben, of Houstonia, Mo., died October 31, 1936.

Born August 16, 1867, at Brooklyn, N. Y., Dr. Hueben entered the service of the U. S. Bureau of Animal Industry as a lay inspector, September 8, 1906. He remained in the service while pursuing his studies at the Kansas City Veterinary College. He was graduated with the class of 1910 and remained in the government service until May 6 of that year. Shortly thereafter, he engaged in the commercial production of anti-hog cholera serum and was one of the pioneers in this industry.

Dr. Hueben was commissioned as second lieutenant in the Veterinary Corps of the Army, August 10, 1917, and about a month later was directed to report to the 87th Division at Camp Pike, Ark. From January 1, 1918, to December 19, 1918, he was on duty inspecting the stockyards at Meridian, Miss. On September 30, 1918, he was promoted to first lieutenant, and on December 20, was assigned to the Auxiliary Remount Depot at Camp Shelby, Miss. He was discharged February 10, 1919. He then located in Houstonia, Mo., and practiced there until his death.

Dr. Hueben joined the A. V. M. A. in 1928. He was a member of the Missouri Veterinary Medical Association. He is survived by his widow, three daughters, two sons and two sisters. One son, Dr. Frank H. Hueben (K. C. V. C. '11), operates a farm near Leavenworth, Kans., in conjunction with his practice.

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**DANIEL J. HICKEY**

Dr. Daniel J. Hickey, of Salt Lake City, Utah, died November 5, 1936. He was born at Albany, N. Y., May 8, 1873, and was a graduate of the U. S. College of Veterinary Surgeons, class of 1910. He was in the service of the U. S. Bureau of Animal Industry from March 6, 1911, until his death, having been assigned to meat inspection and field work at various stations.

Dr. Hickey joined the A. V. M. A. in 1929.

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**GROVER C. STONG**

Dr. Grover C. Stong departed this life at his home in Waterloo, Iowa, on December 1, 1936. His death was the result of a heart attack.

Born June 9, 1884, at Kilbourne, Iowa, Dr. Stong received his early education in the public schools of Van Buren County, and was a graduate of the Keosauqua High School. His veterinary

education was obtained at the Iowa State College. Following his graduation in 1909, he entered private practice in his home county.

On February 9, 1914, Dr. Stong entered the service of the U. S. Bureau of Animal Industry and was assigned to meat inspection at South Omaha, Nebr., where he remained until July 16, 1919, when he was transferred to Des Moines, Iowa. On January 1, 1924, he was transferred to the state force engaged in coöperative tuberculosis eradication work in Iowa. This arrangement continued until May 1, 1928, when Dr. Stong was reappointed to the Bureau and assigned to tuberculosis eradication work at Des Moines. At the time of his death, he was in charge of the work in five Iowa counties.

During the 13 years that Dr. Stong was engaged in the eradication of bovine tuberculosis in Iowa, he worked in the most highly infected areas of the state. A few years ago, it was generally conceded that he had, up to that time, branded more cattle for tuberculosis than any other veterinarian. Of course, as time went on and herds in badly infected areas in other states came under the test, it is possible that other veterinarians may have identified more tuberculin reactors than had Dr. Stong. His interest in this work was exceeded only by his interest in his home and his friends. He had devoted his life wholeheartedly to his wife and two children. Their happiness was his supreme joy.

Dr. Stong joined the A. V. M. A. in 1918.

J. A. B.

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### ALONZO HENRY CHENEY

Dr. A. H. Cheney, of Polson, Mont., died at the Polson Hospital, November 25, 1936, after an illness of but a few hours.

Born in Wisconsin, April 19, 1870, Dr. Cheney moved to California when quite young. He grew to manhood and was graduated from the Stockton Business College. He then studied veterinary medicine at the University of Pennsylvania. Following his graduation in 1904, he went to Montana and was successively located at Miles City, Anaconda and Polson. He practiced at the latter place for 22 years.

Dr. Polson joined the A. V. M. A. in 1909 and for many years was a faithful member of the Montana Veterinary Medical Association in which he held the office of vice-president at the time of his death. He is survived by his widow (née Ruth Sharp) and two sons.

H. M.

### CHARLES F. HARRE

Dr. Charles F. Harre, of McLeansboro, Ill., died suddenly at his home, December 1, 1936, at the age of 57. He had been in poor health for several years. He was a graduate of the Kansas City Veterinary College, class of 1913, and had practiced in southern Illinois for a number of years. Dr. Harre took an active interest in civic affairs of the community, and served two terms on the McLeansboro City Council as alderman from his ward. He is survived by his widow, two sons and one daughter.

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### WILLIAM J. JOHNSON

Dr. William J. Johnson, of Paw Paw, Mich., one of the oldest practicing veterinarians in the state, died at his home on December 11, 1936.

Born at Islington, Ont., June 28, 1857, Dr. Johnson was a graduate of the Ontario Veterinary College, class of 1885. He located in Paw Paw in 1892. At one time he served as a village trustee.

Dr. Johnson was a member of the Michigan State Veterinary Medical Association and was a past president of the Southwestern Michigan Veterinary Medical Association. He was a charter member of the Knights of Pythias lodge of Paw Paw. He is survived by his widow (née Belle Gremps), one daughter, two brothers and one sister.

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### JOHN BERTRAM HAGENBUCH

Dr. J. Bertram Hagenbuch, of Philadelphia, Pa., died in a Pittsburgh hospital, December 11, 1936, at the age of 65 years. He had been taken ill in Butler, Pa., the previous day, and was removed to the hospital. Death was due to heart disease.

A native of Mahanoy City, Pa., Dr. Hagenbuch was graduated from the Philadelphia College of Pharmacy before he entered the University of Pennsylvania for the study of veterinary medicine. Following his graduation in 1895, he opened a drug store in West Philadelphia and conducted this in conjunction with his practice for about 25 years. Dr. Hagenbuch then became associated with the Mulford Biological Laboratories in a sales capacity. Later, he accepted a position as inspector with the U. S. Bureau of Animal Industry, a post he held until his death. He is survived by his widow and one son, Dr. John B. Hagenbuch (U. P. '28), of Lawrenceville, N. J.

A. H. C.

**ARTHUR H. TINGMAN**

Dr. Arthur H. Tingman, of Calumet, Mich., died at the home of his sister, in Mohawk, Mich., December 14, 1936, in his 50th year, after an illness of two months.

Born in Swedetown, Mich., Dr. Tingman spent his entire life in the Calumet district. He was graduated from Ferris Institute, at Big Rapids, before entering the Grand Rapids Veterinary College. Following his graduation in 1915, he went to Minneapolis for a short time. He returned to Calumet and engaged in general practice.

Dr. Tingman was a member of the American Legion and the Calumet Aerie of Eagles. He is survived by his mother and one sister.

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Our sympathy goes out to Dr. E. B. Ackerman, of Huntington Bay Village, N. Y., on the death of his wife, Mrs. Ruth Howe Ackerman, suddenly, November 23, 1936, and to Dr. V. G. Bromwell, of Center Point, Iowa, in the death of his father, Thomas G. Bromwell, in Cedar Rapids, Iowa, December 14, 1936, after a brief illness.

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**PERSONALS**

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**BIRTH**

To DR. and MRS. J. T. BURRISS, of Columbus, Ohio, a son, James Donald, November 28, 1936.

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**PERSONALS**

DR. KENNETH S. JONES (O. S. U. '36) reports a change of address from Kenton to Rushsylvania, Ohio.

DR. P. V. CLARKSON (U. P. '22), formerly located in Lancaster, Pa., gives his new address as Oxford, Pa.

DR. H. H. SEELY (Chi. '10), of Jerseyville, Ill., was elected coroner of Jersey County at the general election on November 3.

DR. L. B. WOLCOTT (K. S. C. '12) recently built a new small-animal hospital at 1434 West Slauson Ave., Los Angeles, Calif.

DR. HUGH HERRON (Ind. '13), of Watseka, Ill., has been reappointed Iroquois County Veterinarian by the Board of Supervisors.

DR. R. W. GROSSMAN (McK. '13), of Columbia, Ill., has been elected Saint Clair County Veterinarian by the Board of Supervisors.

DR. LEONARD E. SWANSON (O. S. U. '27), of Ogden, Utah, is now at the University of Hawaii in the capacity of associate parasitologist.

DR. C. E. DORNHEIM (Chi. '02), of Mount Airy, Md., recently suffered an acute heart attack necessitating confinement to his home for several days.

DR. WILLIAM K. BRECKENRIDGE (O. S. U. '04), of Wheeling, W. Va., was retired from the service of the U. S. Bureau of Animal Industry, effective August 31, 1936.

DR. W. C. GLENNEY (Iowa '36), of Union, Iowa, has purchased the Tri-City Animal Hospital and general practice of Dr. Otto Stader (U. P. '18), at Geneva, Ill.

DR. WESLEY A. YOUNG (Iowa '19), formerly with the Animal Rescue League, of Boston, Mass., is now superintendent of the Anti-Cruelty Society of Chicago, Illinois.

DR. A. E. STETTLER (O. S. U. '16), who has been engaged in Bang's disease control with the Department of Agriculture, has entered private practice at Waynesville, Ohio.

DR. CARROLL P. HART (O. S. U. '06), has been retired from active service in the U. S. Bureau of Animal Industry. He was in charge of meat inspection at Cleveland, Ohio.

DR. FRANKLIN PIERCE (O. S. U. '08) has purchased the veterinary hospital in Wilmette, Ill., formerly owned and operated jointly by himself and the late Dr. Frederick C. Buschbom.

DR. JESSE SAMPSON (Corn. '30), who was an instructor in the New York State Veterinary College for six years following his graduation, has entered general practice at Waterman, Ill.

DR. E. B. SIMONDS (U. S. C. V. S. '18), U. S. B. A. I. inspector-in-charge of tuberculosis eradication in Maryland, will spend January and February in Florida, on a well-earned vacation.

DR. FORREST McCLEAD (O. S. U. '33), who has been on the staff of the Ellin Prince Speyer Animal Hospital in New York City, has taken over the practice of the late Dr. A. E. George, in Flint, Mich.

DR. J. H. BOYD (Ont. '13), of Clayton, Mich., scalded his left hand with a boiling solution of calcium chloride in November. He has recovered from the injury and has returned to practice.

DR. H. M. DeVOLT (Corn. '23), poultry specialist of the Maryland Live Stock Sanitary Service, was operated upon for appendicitis recently and is reported to be making a satisfactory recovery.

DR. FRANCIS M. TIBBET (T. H. '16), formally opened his new hospital at Kingman, Ind., on October 24. Accommodations have been provided for 62 patients. Local newspapers gave Dr. Tibbet some nice publicity.

DR. A. C. JERSTAD (Wash. '33) has left the Western Washington Experiment Station, at Puyallup, and has accepted a position at the Zoölogical Division, U. S. Bureau of Animal Industry, at Beltsville, Md.

DR. F. M. McCONNELL (Gr. Rap. '15) and family, of Litchfield, Mich., started for Florida just before Christmas, to spend several months in the hopes that the warm climate would improve the doctor's health.

DR. EMMETT W. CANTRALL (Wash. '36), formerly assistant to Drs. Massey and McComb, of Santa Barbara, Calif., is now engaged in Bang's disease control work in Washington, on the force of Dr. J. C. Exline.

DR. KARL F. MEYER (Zurich '09), of the Hooper Foundation for Medical Research, San Francisco, Calif., was elected vice-president of the American Society of Tropical Medicine, at the 32nd annual meeting, held in Baltimore, Md., November 18-20, 1936.

DR. MAURICE C. HALL (Geo. Wash. '13), of the National Institute of Health, Washington, D. C., was elected a councilor of the American Academy of Tropical Medicine at the third annual meeting of the organization, in Baltimore, Md., November 18, 1936.